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WAYNE G. BRANDSTADT, *Colonel, MC, U. S. A., Editor-in-Chief*
ROBERT J. BENFORD, *Colonel, U. S. A. F. (MC), Associate Editor*
BENNETT F. AVERY, *Captain, MC, U. S. N., Associate Editor*

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FOREWORD

The *United States Armed Forces Medical Journal* is the medium for disseminating information of administrative and professional interest to all medical personnel of the Department of Defense. The Chairman of the Armed Forces Medical Policy Council and the Surgeons General of the several services invite all medical officers, dental officers, Medical Service Corps officers, Nurse Corps officers, and officers of the Veterinary Corps of the Armed Forces, and the medical consultants of the Army, Navy, and Air Force to submit manuscripts for publication in this *Journal.*

MELVIN A. CASBERG, M. D.,
Chairman, Armed Forces Medical Policy Council,
Department of Defense.

GEORGE E. ARMSTRONG,
Major General, U. S. A.,
Surgeon General, U. S. Army.

LAMONT PUGH,
Rear Admiral, MC, U. S. N.,
Surgeon General, U. S. Navy.

HARRY G. ARMSTRONG,
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Address: Editor, U. S. Armed Forces Medical Journal, Armed Forces Medical Publication Agency, 23d and E Streets NW., Washington 25, D. C.

WAYNE G. BRANDSTADT, *Editor-in-Chief,*
Colonel, Medical Corps,
United States Army.

ROBERT J. BENFORD, *Associate Editor,*
Colonel, Medical Corps,
United States Air Force.

BENNETT F. AVERY, *Associate Editor,*
Captain, Medical Corps,
United States Navy.

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Monthly Message

"The past years of combat experience have taught me the absolute necessity of competent and adequate medical support for the success of a military mission." These were the words of General Ridgway as he addressed a special session of the top medical military leaders of the NATO countries recently gathered at SHAPE, and continuing in his role as Supreme Allied Commander Europe, the General pledged his support to any realistic program developed to strengthen the medical organizations under his command.

Not only was the immediate beneficial psychologic impact of such a supportive statement quite evident, but equally obvious was the fact that the effects of such an assurance would be observed for some time to come as measured by unified efforts to resolve our common problems. Though the three short days of the session barely permitted an appreciation of the numerous tasks awaiting solution, this realization in itself afforded a definite stimulus for future progress. The necessity for coordination and a certain degree of uniformity in basic field medical activities was evident as exemplified by requirements for uniform field emergency medical tags, narcotic dosages, stretchers, immunizations, and other related items.

The United States was represented by the Chairman and members of the Armed Forces Medical Policy Council. Besides the orientation briefings and technical discussions, papers covering such subjects as the correct use of blood derivatives, forward combat medical care, and methods of evacuation of the wounded were presented by delegates from the United Kingdom, France, and the United States.

The personnel of the medical division of SHAPE under the direction of Brig. Gen. William Kennard are to be complimented for accomplishing a difficult organizational task in a most efficient manner.

Melvin A. Casberg

Melvin A. Casberg, *M. D., Chairman*
Armed Forces Medical Policy Council
Office of the Secretary of Defense

ACUTE RENAL INSUFFICIENCY

PAUL D. DOOLAN, *Lieutenant, MC, U. S. N. (1)*

ACUTE renal insufficiency is variously known as lower nephron nephrosis, crush syndrome, acute urinary supression, hemoglobinuric nephrosis, traumatic anuria, and necrotizing nephrosis. All of these descriptive titles have reference to a temporary inadequacy of the excretory and metabolic functions of the kidney which follows a variety of causes. Inadequacy of excretory function is the problem of immediate concern and may be defined as the inability of the body to produce a urine of sufficient volume and/or concentration to prevent the retention of metabolites. For the average adult a minimum of from 300 to 400 cc. of maximally concentrated urine will preserve homeostasis, but this value will vary depending on the energy metabolism of the body and the ability of the renal tubules to do osmotic work.

ETIOLOGY

The causative agents are so numerous that the clinician may find it helpful to think in terms of etiologic groups even though this leads to some oversimplification. They cannot be rigidly differentiated and in many patients several causes are demonstrable. The groups are: (1) shock resulting from hemorrhage, trauma, severe dehydration, or any other cause; (2) tissue destruction resulting from crushing injuries, burns, hemolytic reactions, premature separation of the placenta, et cetera; (3) cytologic poisons such as bichloride of mercury, carbon tetrachloride, uranium, oxalates, sulfonamides, et cetera; and (4) bilateral cortical necrosis of the kidney caused by a variety of agents and perhaps of a separate and distinct pathogenesis. (Metabolic alkalosis may seriously impair renal function but whether it should be added as a fifth group is debatable.)

A thorough knowledge of the underlying pathologic physiology is vitally necessary in the management of patients with acute renal insufficiency. Accordingly the salient concepts relative to the etiologic agents, the kidney, cellular metabolism, and the insensible water loss will be presented in some detail.

PATHOLOGIC PHYSIOLOGY: THE ETIOLOGIC AGENTS

Group 1. The kidney reacts poorly to blood and oxygen deprivation: (1) because glomerular filtration requires an arterial pressure

(1) Formerly Senior Assistant Resident in Medicine and Fellow in the Department of Metabolism and Endocrinology, Georgetown University Hospital, Washington, D. C.

of from 60 to 70 mm. of mercury and any fall to levels less than these causes a cessation of filtrate formation; (2) more important, whenever systemic hypotension occurs a vasoconstriction of the renal vessels takes place and blood is shunted away from the kidneys to more vital organs such as the brain and heart *(2-4)* (whether humoral or neural stimuli are responsible for this reaction is not known but the result is the same in that a marked decrease in renal blood flow occurs); and (3) coincidentally with this ischemia, hypoxia of the kidney tissue develops. Normally, about 1,500 cc. of blood flows through the kidneys each minute, from each 100 cc. of which the parenchyma extracts from 0.5 to 2 volumes of oxygen. Despite this low extraction ratio the total oxygen consumption lies in the neighborhood of from 10 to 20 cc. per minute because of the large total volume of blood traversing the kidney each minute *(5)*. Therefore any substantial reduction in the renal blood flow ordinarily results in hypoxia. An additional encumbrance in renal defense has been demonstrated *(2-4)*. When hypotensive patients are transfused sufficiently to restore the systemic blood pressure and cardiac output to within normal limits, the renal blood flow remains subnormal and a lag period ensues before normal values are achieved. Thus hypotension is responsible for kidney damage because of cessation of glomerular filtration, vasoconstriction, and hypoxia.

Group 2. Whenever tissue is destroyed or rendered ischemic a variety of toxins and pigmented compounds are liberated into the circulation *(4, 6, 7)*. The substances of chief importance appear to be hemoglobin, the oxygen carrier of the blood, and myoglobin, the oxygen container of muscle. The myoglobin molecule is only ¼ as large as that of hemoglobin and has been found to filter 25 times as rapidly *(8)*. Because its structure closely resembles that of hemoglobin it is capable of giving a positive test for blood when excreted in the urine, and in such cases identification must be made by spectrographic analysis. These compounds will not cause kidney damage unless they are precipitated within the tubule and this requires an acidic and con-

(2) Lauson, H. D.; Bradley, S. E.; and Cournand, A.: Renal circulation in shock. J. Clin. Investigation 23: 381-402, May 1944.

(3) Selkurt, E. E.: Renal blood flow and renal clearance during hemorrhage shock. Am. J. Physiol. 145: 699-709, Mar. 1946.

(4) Corcoran, A. C., and Page, I. H.: Crush syndrome; post-traumatic anuria; observations on genesis and treatment. J. A. M. A. 134: 436-441, May 31, 1947.

(5) Cargill, W. H., and Hickam, J. B.: Oxygen consumption of normal and diseased human kidney. J. Clin. Investigation 28: 526-532, May 1949.

(6) Bywaters, E. G. L.: Ischemic muscle necrosis; crushing injury, traumatic edema, crush syndrome, traumatic anuria, compression syndrome; type of injury seen in air raid casualties following burial beneath debris. J. A. M. A. 124: 1103-1109, Apr. 15, 1944.

(7) Bywaters, E. G. L., and Beall, D.: Crush injuries with impairment of renal function. Brit. M. J. 1: 427-432, Mar. 22, 1941.

(8) Yuile, C. L., and Clark, W. F.: Myohemoglobinuria; study of renal clearance of myohemoglobin in dogs. J. Exper. Med. 74: 187-196, Sept. 1941.

centrated tubular fluid. (The patient's state of hydration is also important. Dehydration favors precipitation.) The mechanical blockage that results impairs the function of the involved and adjacent nephrons. Hemoglobin and myoglobin are not directly injurious to renal tissue but their degradation products which arise after precipitation in the tubules can give rise to acid hematin which is a definite cytotoxic poison. It is a debated point as to whether these substances, per se, are capable of causing acute renal failure and most patients encountered bear evidence of an additional factor such as a period of shock. Their presence, in addition, undoubtedly results in more severe damage than that accompanying shock alone. Thus tissue destruction is responsible for some degree of kidney damage because of liberation of pigmented compounds capable of mechanically blocking and chemically injuring the renal tubules.

Group 3. The cytologic poisons cause renal failure by a direct action on the kidney parenchyma. The inflammatory response elicited potentiates the damage and prolongs recovery by interfering with the circulation and oxygenation of the involved and adjacent tissue.

Group 4. Bilateral cortical necrosis of the kidneys is an unusual cause of acute renal insufficiency most often encountered in pregnancy but also in men and children *(9-11)*. Although most patients with this condition have died, with the use of newer methods of treatment, some such patients have survived. This condition can be produced experimentally by the injection of certain toxins which cause vasoconstriction and thrombus formation in the smaller arteries and arterioles of the renal cortex. This intense vasoconstriction may be due to a mechanism similar to that described by Trueta et al. *(12)*.

The English investigators described a mechanism whereby, after application of a tourniquet to the hind leg of a rabbit, the renal flow would remain normal in amount but would traverse a completely different course. The basis of this rerouting devolves on the juxtamedullary glomeruli whose efferent branches drain directly into larger venous channels without the intermediation of a capillary bed such as occurs in all other cortical circuits. Under the above experimental circumstances cortical vasoconstrictions shunts all of the blood into the veins via the patent juxta-

(9) Duff, G. L., and More, R. H.: Bilateral cortical necrosis of kidneys. Am. J. M. Sc. 201: 428-450, Mar. 1941.

(10) Haft, D. E., and Prior, J. T.: Bilateral cortical necrosis of kidneys following treatment of an unusual case of heart block. Ann. Int. Med. 34: 1483-1491, June 1951.

(11) Kimmelstiel, P.: Vascular occlusion and ischemic infarction in sickle cell disease. Am. J. M. Sc. 216: 11-19, July 1948.

(12) Trueta, J.; Barclay, A. E.; Daniel, P. M.; Franklin, K. J.; and Pritchard, M. M. L.: Studies of the Renal Circulation. Charles C Thomas, Publisher, Springfield, Ill., 1947.

medullary glomeruli and the resulting cortical ischemia is the cause for the subsequent renal failure. Other investigators have acknowledged the importance of vasoconstriction in the genesis of acute kidney failure but do not believe it occurs in such a manner. Direct and indirect measures of the renal blood flow have shown it to be markedly reduced rather than normal in amount as Trueta found *(2–4, 13, 14)*.

Studies on the oxygen extraction ratio have yielded normal values, but if shunting were operative the blood should perfuse only the metabolically inactive medulla and should not be using a normal percent of oxygen *(14–16)*. Other investigators have been unable to reproduce the Oxford group's results when experimental animals other than the rabbit were used *(16, 17)*. Thus the preponderance of evidence is against the importance of bypasses in the pathogenesis of acute renal insufficiency. Whether bilateral cortical necrosis of the kidney is an exception to this remains to be proved.

PATHOLOGIC PHYSIOLOGY: THE KIDNEY

Once the damage has been sustained by the renal parenchyma the urine volume abruptly declines. The exact cause of the oliguria is unknown but at least 3 factors are operative: (1) back diffusion of the filtrate across the tubular epithelium; (2) functional ischemia of the cells; and (3) mechanical blockage of the tubules by casts. The role played by each of these factors, singly or in combination, varies with the agent responsible for the renal failure. A comprehensive review of this subject has been made by Smith *(18)*.

Regeneration of the tubular epithelium begins soon after the injury and continues in such a fashion that back diffusion of the filtrate is progressively curtailed. Concomitantly, the renal blood flow and glomerular filtration rate improve and these changes are witnessed by the passage of increasingly larger volumes of urine. This should be viewed only as an encouraging sign for the tubular epithelium has by no means regained its normal

(13) Sirota, J. H.: Carbon tetrachloride poisoning in man; mechanisms of renal failure and recovery. J. Clin. Investigation 28: 1412-1422, Nov. (pt. 2) 1949.

(14) Moyer, J. H., and Greene, J. A.: Further studies on existence of renal bypasses. Am. J. Med. 9: 280, 1950.

(15) Clark, J. K.; Barker, H. G.; and Crosley, A. P., Jr.: Evidence against renal vascular shunts in case of lower nephron nephrosis. Am. J. Med. 9: 268-271, Aug. 1950.

(16) Moyer, J. H.; Conn, H. L., Jr.; Markley, K.; and Schmidt, C. F.: Hemodynamics of renal circulation. Am. J. Physiol. 159: 582, 1949.

(17) Study, R. S., and Shipley, R. E.: Effects of renal artery-nerve stimulation on renal blood flow measured directly and by clearance and extraction of diodrast. Am. J. Physiol. 159: 592, 1949.

(18) Smith, H. W.: Kidney; Structure and Function in Health and Disease. Oxford University Press, New York, N. Y., 1951.

faculties of selective reabsorption and the renal hemodynamics are still far from normal. For instance, if tubular reabsorption is poor, as much as 5,000 cc. of urine may be excreted in 24 hours when the glomerular filtration rate is less than 5 percent of normal. Although clearance measurements during the early phases of diuresis have been found to be extremely low, it should be remembered that their absolute values bear no correlation with the ultimate outcome of the disease *(13)*. In addition, during this same period, the tubules apparently deal normally with the chloride ion even though other transport mechanisms remain severely impaired.

From 7 to 9 days after diuresis begins the clearance values rapidly improve and continue to do so during the next 5 to 7 weeks but thereafter improvement takes place more moderately and normal values are arrived at by the sixth or seventh month. Studies to date have failed to show any consistent pattern in the time that all renal function tests will have reverted to normal but the ability to form a concentrated and strongly acidic urine are usually among the last to recover.

PATHOLOGIC PHYSIOLOGY: CELLULAR METABOLISM AND THE INSENSIBLE WATER LOSS

Coincidentally with the changes which take place within the kidney, normal cellular catabolism liberates 3 important groups of substances into the extracellular fluid: (1) the end products of metabolism including organic acids, (2) osmotically potent electrolytes, and (3) the molecularly contained water. The end products of protein metabolism are of major consequence to the oliguric patient because they demand renal excretion while the carbon dioxide produced by the oxidation of carbohydrate and fat is readily expired. Although ketosis can be prevented by supplying sufficient carbohydrate the accumulation of other organic acids cannot be so easily controlled.

Osmotically potent electrolytes also gain egress from the cells as a result of catabolic processes. The ion of gravest interest being potassium whose chief route of elimination from the body is via the kidney. The intracellular concentration of potassium is about 30 times as great as that found in the extracellular fluid and this gradient differential in the presence of ablated renal function facilitates its accumulation in the extracellular fluid where excessive concentrations of potassium are fatal. Cellular catabolism also affects the body fluids directly. The oxidation of carbohydrates, protein, and fat yields water, the so-called water of oxidation, and the average adult acquires about 250 cc. (3.1 cc. per kg. of body weight) in this way each day.

This chain of events is responsible for the azotemia and metabolic acidosis which develops in the oliguric patient and in addition sets the stage for the poorly understood electrolyte shifts and distortions which take place in the various body fluid compartments. The severity of many features which the patient presents are the direct result of the rapidity with which cellular destruction has occurred and rational management is, therefore, aimed at reducing endogenous catabolism to a minimum.

In addition to the changes which take place within the kidney and the cells and fluids of the body, the concomitant insensible water loss is important, because it becomes of critical significance in the oliguric patient and in the absence of diarrhea, vomiting, and visible perspiration is the only path by which fluid leaves the body. Because the evaporation of 1 gram of water requires 0.58 calories, the vaporization of water from the skin and lung surfaces is a means for the disposal of heat, and the total amount lost in this manner per day is an independent function of the energy metabolism of the body. The fluid lost is devoid of electrolytes. The precise amount expended by a normal man weighing 70 kg. varies between 840 and 1,090 cc. per day depending on the respiratory quotient, the environmental temperature, humidity, and air currents as well as the type and amount of clothing worn *(19–21)*.

Strauss *(22)* called attention to the fact that liver damage also occurs in patients with acute renal insufficiency. Other than in carbon tetrachloride poisonings and in one patient with bichloride of mercury intoxication we have made no special studies of this problem.

PATHOLOGY

The pathologic changes which take place in the kidney have been well described *(7, 23, 24)*. Certain features vary depending on the etiologic agent but salient characteristics are common to all types of acute renal insufficiency. Grossly, the kidneys are swollen, tense, and weigh more than the normal 300 grams. The pale cortex and dusty-looking medulla produces an appearance

(19) Newburgh, L. H., and Johnston, M. W.: Insensible loss of water. Physiol. Rev. 22: 1-18, Jan. 1942.

(20) Gamble, J. L.: Physiological information gained from studies on life raft ration. Harvey Lect. (1946-1947) 42: 247-273, 1947.

(21) Gamble, J. L.: Chemical Anatomy, Physiology and Pathology of Extracellular Fluid. 5th edition. Harvard University Press, Cambridge, Mass., 1947.

(22) Strauss, M. B.: Acute renal insufficiency due to lower-nephron nephrosis. New England J. Med. 239: 693-700, Nov. 4, 1948.

(23) Lucke, B.: Lower nephron nephrosis (renal lesions of crush syndrome, of burns, transfusions, and other conditions affecting lower segments of nephrons). Mil. Surgeon 99: 371-396, Nov. 1946.

(24) Muirhead, E. E.: Acute renal insufficiency. Medical Basic Sciences Course, Army Medical Center Graduate School, Washington, D. C., 1949.

of increased corticomedullary demarcation. Microscopically, changes are seen in the epithelium within from 18 to 24 hours after trauma. These may progress so that eventually all gradations of injury, including necrosis of some cells, are visible. Usually the involvement is patchy in distribution. Sometimes the changes are more severe in the proximal convoluted tubule but usually the distal tubule bears the brunt of the injury. Pigmented casts appear within from 36 to 72 hours. Inflammatory reaction and edema are visible in the interstitial tissue and at times numerous tubulovascular anastomoses may be present. Occasionally, glomerular changes and hypertrophy of the juxtaglomerular apparatus are demonstrable. The tubular epithelium begins to regenerate by about the fourth day and is fairly complete within from 2 to 4 weeks. Rarely, flat films of the abdomen reveal necrotic epithelium which has undergone calcification lying in the lumen of the tubules.

The specific lesions seen in bilateral cortical necrosis include widespread ischemic necrosis of the cortex which may be patchy in distribution or include the entire cortex as well as the columns of Bertin. In the areas of necrosis there is extensive thrombosis of the smaller arteries.

CLINICAL FEATURES

The clinical course of this syndrome varies with the precipitating cause and basic illness. Most patients who succumb do so as a direct result of the basic illness, and before uremia becomes a problem. Ordinarily, anuria or oliguria results within 24 hours after the damage has been sustained. The anuria is transient, being measured in hours and virtually all patients have an oliguria. Early in the course the urine is usually acid, has a specific gravity of from 1.009 to 1.014 (occasionally higher), and is smoky or cloudy in appearance. Sometimes it gives a positive test for occult blood, and pigment may continue to be passed for 3 or 4 days but then this clears completely. This early urine contains a great deal of albumin; granular, cellular, and broad renal-failure casts; numerous cells; and amorphous minerals. As more urine is passed, the amount of sediment progressively decreases and the specific gravity remains fixed at about 1.010.

In the first 5 or 6 days there is a gradually progressive elevation of the serum urea nitrogen, nonprotein nitrogen, phosphates, and sulfates. The serum creatinine rises rapidly at first and it is not until the second week that smaller increments of ascent are noted. The serum sodium and chloride usually fall in a parallel manner, at first gradually, but then more rapidly with the fall in chloride being proportionally greater. Concomitantly the carbon dioxide content falls and acidosis increases in

severity. During this first period the patient may continue to feel well but as the second week begins a progressive apathy usually attended by nausea and vomiting develops. This may continue until there is almost continual somnolence and finally severe uremia with coma. There is no correlation between the severity of uremia and the eventual outcome.

All patients who do not have diarrhea and who remain oliguric long enough, develop potassium intoxication. This may be rapidly lethal and because effective means of treatment are available, early detection is important. The electrocardiographic changes which appear earliest in intoxication include: (1) an increase of height and pointing of the T waves, then slanting of the S-T segment followed by prolongation of the P-R interval, and later disappearance of the P waves; (2) as the poisoning continues the QRS complexes become prolonged, the R wave is lowered, and eventually the QT interval is markedly prolonged; and (3) terminally, arrhythmias develop and finally asytole. There is no correlation between the electrocardiographic changes and the height of the serum potassium so that both determinations should be performed. The neurologic changes occur later and are the same for both hyperkalemia and hypokalemia. There is areflexia and flaccid paralysis which may be of an ascending type. Aphonia and respiratory distress occur late. Paresthesias occur in patients with hypokalemia more frequently than in those with hyperkalemia. Intoxication can occur before uremia becomes well marked and a low serum sodium level facilitates the development of potassium intoxication.

There is no uniformity to the precise acid-base and fluid-balance derangements that develop in different patients. This is due, in large measure, to iatrogenic causes. Despite repeated warnings excess fluid and base continue to be given to a large number of these patients. Because pulmonary edema is still one of the most frequent causes of death, this procedure cannot be condemned too harshly. In other patients base has been omitted but water administered in excess. These patients, especially if the serum sodium was low initially, may develop water intoxication. If this condition progresses, the serum osmolarity falls, the spinal fluid pressure rises, and convulsions occur. This demands immediate treatment with hypertonic saline and carries with it the risk of precipitating acute pulmonary edema.

Periorbital edema is sometimes seen in oliguric patients in the absence of peripheral edema. Some elevation of blood pressure occurs in almost all patients and usually is of a mild form. The hypertension may appear as early as the third day of oliguria or not until diuresis has become well established. Convulsions may occur in the absence of hypertension.

Diuresis ordinarily begins between the seventh and fourteenth days but may not appear until later. Diuresis may appear late in patients with chronic renal disease in whom acute renal insufficiency is superimposed. Usually the uremia does not attain its meridian until from 4 to 9 days after diuresis has commenced. Once large urine volumes are achieved the danger is by no means over. Pulmonary edema may still result from overzealous administration of fluids. Water intoxication or salt depletion states develop because of failure to replace the electrolytes lost in the copius urine. Dehydration may result from inadequate fluid replacement because of failure to appreciate that the insensible water loss continues and that the kidneys do not regain their capacity to concentrate until later. Potassium deficiency is frequently encountered.

In addition to these, another distressing complication may occur. Luetscher and Blackman *(25)* noted a marked elevation of serum sodium and chloride associated with severe brain damage during the diuretic phase in patients with acute renal insufficiency resulting from sulfathiazole. Borst *(26)* also observed marked elevation of the serum chloride and hyperazotemia associated with low to absent urinary sodium chloride in patients with gastrointestinal hemorrhages. Excessive tubular reabsorption of sodium and chloride during this phase may occur in acute renal insufficiency from other causes. Little can be done in the way of treatment other than restriction of salt intake although Merrill *(27)* has successfully treated such patients with an artificial kidney.

THERAPY

Because acute renal insufficiency presents a succession of developments and problems, therapy must be adaptable in character and well founded in principle. Some simple prophylactic measures may prevent the development of this condition. Dehydrated and anemic patients should be thoroughly prepared to withstand any stress. Shock must be detected early and treated vigorously. Specific antidotes such as BAL should be administered as soon as possible after poisoning. Precautionary measures must be enforced with all intravenous infusions, and in transurethral prostatic resections the wound should not be irrigated with distilled water. In hemolytic reactions, mercury intoxication, and other causes of oliguria, it is wise to insert a bladder

(25) Luetscher, J. A., Jr., and Blackman, S. S., Jr.: Severe injury to kidneys and brain following sulfathiazole administration; high serum sodium and chloride levels and persistent cerebral damage. Ann. Int. Med. 18: 741-756, May 1943.

(26) Borst, J. G. G.: Cause of hyperchloremia and hyperazotemia in patients with recurrent massive hemorrhage from peptic ulcer. Acta med. Scandinav. 97: 68-88, 1938.

(27) Merrill, J. P.: Artificial kidney. New England J. Med. 246: 17-27, Jan. 3, 1952.

catheter and measure the urine collections hourly. In this manner the fear of anuria will not prevent proper hydration therapy; acute renal insufficiency may be prevented, and if not, its presence will be detected early.

The following outline and generalizations describe the conservative management of the oliguric phase.

1. Intake and output records should be accurate to the cubic centimeter.

2. Body weight measurements should be made using the same scale at the same time each day.

3. The daily fluid intake should be limited to the replacement of the insensible water loss in addition to the total amount of urine, stool, and vomitus collected.

4. At least 100 grams of carbohydrate should be provided daily.

5. Daily or every other day, the following laboratory determinations should be performed: hematocrit, serum urea nitrogen, carbon dioxide combining power, chloride, sodium, and potassium. Less frequently needed are: total serum proteins, calcium, phosphorous, and creatinine. Serial determinations of the serum osmolarity, if available, are of distinct value.

6. An ECG should be taken every other day.

7. Prophylactic penicillin or proper antibiotic therapy should be instituted.

8. Vitamins (including fat soluble) are routinely prescribed.

The necessity of keeping precise intake and output records cannot be overemphasized. Errors are simple to make even under optimal clinical conditions and in an insidious manner produce serious complications. For instance, infusions do not contain exactly 1,000 cc. as many physicians assume, but usually about 115 cc. in excess of this. Over a period of several days the accumulated excess may cause congestive heart failure.

As a valuable adjunct to fluid balance records in guiding therapy and because it frequently is impossible to estimate the amount of vomitus or liquid stools passed, all patients should be weighed daily on scales accurate to at least 250 grams. Stretchers and special scales are available for weighing bed ridden patients.

The insensible water loss is adequately replaced, in the clinical sense, by giving 0.5 cc. per kg. of body weight per hour *(20, 21)*. Electrolyte determinations performed on aliquots of the urine, vomitus, and fecal material aid in replacing those lost. Equivalent volumes of fluid are used to replace those

passed. If facilities are not available for daily electrolyte determinations, pH values by means of nitrazine paper should be made on all fluids eliminated.

Acidosis invariably develops and it is difficult to treat because of ionic shifts occurring between the extra- and intracellular compartments in addition to other poorly understood phenomena. Because treatment is fraught with the hazard of producing congestive heart failure the clinician should proceed cautiously. At times, hypertonic solutions of base in small volumes given slowly are of distinct value.

Minimizing cellular catabolism inhibits endogenous protein breakdown, the progression of azotemia, acidosis, and potassium intoxication, as well as distributional changes in the body fluids. Such a minimizing of cellular breakdown can be effectively accomplished in the normal human subject by the daily provision of 100 grams (400 calories) of glucose *(20, 21)*. This quantity has been accepted by many as the optimal amount to be given to the oliguric patient. Others have stated that more salutary results are accomplished by providing from 1,700 to 2,500 calories in the form of carbohydrate and fat *(18, 28–30)*. Analysis of Butler's *(31)* own data would seem to corroborate this.

I have used mixtures containing from 800 to 1,800 calories in the form of glucose and oral fat emulsion, given slowly by intragastric drip. Testosterone and lipotropic agents were administered concomitantly. These substances were originally recommended to me by Merrill. Infrequently, this method of administration was not tolerated or was contraindicated and in such instances, at least 400 calories were provided by vein. In such patients the use of plastic venous catheters has proved to be of material help.

The treatment of potassium intoxication has been admirably studied by Merrill et al. *(32)*. Experience with the modified Kolff artificial kidney has led me to believe that it is the most rapid and effective means of treatment. Hemodialysis has consistently produced satisfactory results of a more permanent nature than have been obtained by any other means. Treatment

(28) Borst, J. G. G.: Protein katabolism in uraemia; effects of protein-free diet, infections, and blood-transfusions. Lancet 1: 824-828, May 29, 1948.

(29) Bull, G. M.; Jockes, A. M.; and Lowe, K. G.: Conservative treatment of anuric uraemia. Lancet 2: 229-234, Aug. 6, 1949.

(30) Snapper, I.: Management of acute renal failure. Bull. New York Acad. Med. 25: 199, 1949.

(31) Butler, A. M.: Minutes of the Conference on Metabolic Aspects of Convalescence Including Bone and Wound Healing. 8th meeting, Josiah Macy, Jr. Foundation, New York, N. Y., Oct. 13-14, 1944. p. 177.

(32) Merrill, J. P.; Levine, H. D.; Somerville, W.; and Smith, S., III: Clinical recognition and treatment of acute potassium intoxication. Ann. Int. Med. 33: 797-830, Oct. 1950.

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by means of resins and intermittent peritoneal lavage hold some promise but require further study. Calcium, hypertonic saline solution, glucose and insulin, DUCA, and gastrointestinal lavage have not proved to be satisfactory methods of treatment.

One should not be led to believe that acute renal insufficiency will run a self limited course and that all that is needed is the above schema of conservative management. The general features presented by a healthy young man who accidentally inhaled carbon tetrachloride and the elderly postoperative patient are entirely different even though both have acute renal insufficiency. Major problems of interplay between the basic illness, the complicating conditions, and the oliguria are ever present. Some such problems in the oliguric patient are congestive heart failure, reoccurrence of shock, severe diarrhea or gastrointestinal hemorrhage, infection, high fever, and the problems of drug dosages and reactions which may arise with digitalis, antibiotics, BAL, et cetera. In patients particularly difficult to treat and those with potassium intoxication, other modalities in addition to conservative therapy have proved to be lifesaving. Of these other therapeutic modalities the following are the most important *(27)*.

Artificial kidney. Many types have been developed *(33–37)*. The modified Kolff model described by Merrill et al. *(38, 39)* has been used extensively with excellent results. It rapidly and effectively removes metabolites and corrects acidosis in addition to being the treatment par excellence for potassium intoxication. It removes or greatly improves the signs and symptoms of uremia thereby facilitating clinical management. In some instances pulmonary edema may be cleared and the blood pressure beneficially raised by dialysis. Its disadvantages are that a specially trained physician or team as well as rather elaborate hospital and laboratory facilities must be available in order to operate it safely and effectively. Its use is therefore limited to the larger medical institutions.

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(33) Kolff, W. J.: New Ways of Treating Uraemia. J. & A. Churchill, Ltd., London, 1947.

(34) Murray, G.: Development of artificial kidney; experimental and clinical experiences. Arch. Surg. 55: 505-522, Nov. 1947.

(35) Alwall, N.: On artificial kidney; apparatus for dialysis of blood in vivo. Acta med. Scandinav. 128: 317-325, 1947.

(36) Skeggs, L. T., Jr., and Leonards, J. R.: Studies on artificial kidney; preliminary results with new type of continuous dialyzer. Science 108: 212-213, Aug. 27, 1948.

(37) Kolff, W. J.: Artificial kidney; treatment of acute and chronic uremia. Cleveland Clin. Quart. 17: 216-228, Oct. 1950.

(38) Merrill, J. P.; Thorn, G. W.; Walter, C. W.; Callahan, E. J., III; and Smith, L. H., Jr.: Use of artificial kidney; technique. J. Clin. Investigation 29: 412-424, Apr. 1950.

(39) Merrill, J. P.; Smith, S., III; Callahan, E. J., III; and Thorn, G. W.: Use of artificial kidney; clinical experience. J. Clin. Investigation 29: 425-438, Apr. 1950.

The conventional form of *peritoneal dialysis* has usually proved to be a slow, prolonged, and cumbersome method of treating uremia that carries with it the risk of infection and fluid retention *(40)*. As a consequence its value is questionable. The modification of intermittent peritoneal irrigation described by Grollman et al. *(41)* may prove to be more effective and practical but requires further investigation.

Replacement transfusion is a method of removing metabolites that has been used by Dausset *(42)* and others and which is sometimes combined with peritoneal irrigation. The large volumes of fresh blood required and the dangers of homologous serum jaundice, hemolytic reactions, and congestive heart failure weighed against the moderate amounts of urea removed by repeated replacement transfusions fail to justify this method.

Because the mucosa of the gastrointestinal tract is not a simple dialyzing membrane *gastrointestinal irrigations* have not proved to be of any substantial aid in the management of the oliguric patient. The method is not a very effective means of clearing azotemia and it is particularly difficult to control the fluid and electrolyte balance of patients being treated with either intestinal or colonic irrigations. Perfusion of an isolated intestinal loop can be more accurately controlled and as many as 8.6 grams of urea have been removed by from 8 to 10 hours of this procedure *(43)*. It is difficult to imagine the employment of such a procedure in acute renal insufficiency.

Hoffman and Marshall *(44)* have cautiously induced dependent edema as a means of diluting the products of retention, this *overhydration* being justified by the fact that edema is less injurious than uremia. This method is dangerous and should not be used in the oliguric patient for there is no certainty that pulmonary edema can be avoided. Perhaps in certain patients whose uremia is severe and continuing to progress even though diuresis has commenced, this method may be beneficially employed. Despite the fact that congestive heart failure may still occur, it can be more effectively treated once diuresis has begun

(40) Odel, H. M.; Ferris, D. O.; and Power, M. H.: Peritoneal lavage as effective means of extrarenal excretion; clinical appraisal. Am. J. Med. 9: 63-77, July 1950.

(41) Grollman, A.; Turner, L. B.; and McLean, J. A.: Intermittent peritoneal lavage in nephrectomized dogs and its application to human being. Arch. Int. Med. 87: 379-390, Mar. 1951.

(42) Dausset, J.: Lower nephron nephrosis: report of treatment of 44 patients by repeated replacement transfusions. Arch. Int. Med. 85: 416-431, Mar. 1950.

(43) Twiss, E. E., and Kolff, W. J.: Treatment of uremia by perfusion of an isolated intestinal loop. J. A. M. A. 146: 1019-1022, July 14, 1951.

(44) Hoffman, W. S., and Marshall, D.: Management of lower nephron nephrosis. Arch. Int. Med. 83: 249, 1949.

and the danger of the procedure is thereby lessened. We (45) employed forced dilution to lower the serum nonprotein nitrogen in such a patient and obtained encouraging results.

SUMMARY

Acute renal insufficiency may be caused by shock, tissue destruction, cytotoxic poisons, or bilateral cortical necrosis of the kidneys. The fundamental processes responsible for this condition are renal ischemia and hypoxia, tubular obstruction, and direct cytotoxic destruction of the tubular epithelium.

Oliguria is the result of the back diffusion of the filtrate, functional ischemia of the cells, and mechanical blockage of the tubules by casts. Diuresis serves only as an indication of incipient recovery because large volumes of urine may be passed even though the renal hemodynamics remain severely impaired. Full functional recovery requires about 6 months. Once oliguria supervenes the events taking place within the cells are important. As a direct result of these events, azotemia, acidosis, potassium intoxication, and distortions in the fluid compartments occur. The so-called insensible water loss is of critical significance in the oliguric patient. The amount of fluid lost in this manner is independent of the fluid balance of the body and the status of renal function. Fluid therapy should therefore always include adequate replacement of this expenditure.

There are no consistent pathologic findings, but the injury is usually patchy in distribution, generally maximal in the distal tubular segments, and inflammatory changes with edema are present in the interstitial tissues. Regeneration usually begins by about the fourth day and is fairly complete by the twenty eighth.

The clinical course varies with the precipitating cause and basic illness. Most fatalities are attributable to these factors and occur before uremia becomes a major problem. As oliguria persists, azotemia and metabolic acidosis become progressively more severe. As long as an inadequate volume of urine is passed, potassium intoxication remains a hazard. ECG's must be taken frequently. Distortions of the body fluids frequently occur in the form of peripheral and pulmonary edema and hypertension. After diuresis is established the development of salt and water depletion and hypokalemia should be prevented.

Therapy should be, first of all, prophylactic. During the oliguric phase the fluid balance and body weight records must be accurately kept. Fluid therapy should be limited to replacement

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of the daily losses. Minimizing endogenous cellular catabolism by supplying an adequate caloric intake is important. Acidosis must be treated cautiously.

The artificial kidney is the most effective means available for treating potassium intoxication, clearing uremia, and correcting acidosis.

ADDENDUM. Since this manuscript was submitted, an article on the pathogenesis and pathology of acute renal failure has been published by Oliver, J.; MacDowell, M.; and Tracy, A.: Pathogenesis of acute renal failure associated with traumatic and toxic injury; renal ischemia, nephrotoxic damage and ischemuric episode. J. Clin. Investigation 30: 1307-1439, Dec. 1951, in which they demonstrated the patchy distribution of "tubulorhectic" lesions throughout all parts of a scattered and variable number of nephrons resulting from renal ischemia. They also demonstrated that the lesions following ingestion of nephrotoxins was a necrosis without rhexis which equally involved the functionally concerned segment of all nephrons, and that casts, per se, were not directly injurious to the tubular epithelium.

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Physiology of the Eye. Volume II. Vision, by *Arthur Linksz,* M. D., F. A. C. S., Assistant Clinical Professor of Ophthalmology, New York University, Postgraduate School of Medicine; Associate Attending Surgeon, Manhattan Eye, Ear and Throat Hospital, New York, N. Y. 869 pages; illustrated. Grune & Stratton, Inc., New York, N. Y., publishers, 1952. Price $19.

Kitchen Strategy, by *Leona M. Bayer,* M. D., Assistant Clinical Professor of Medicine, Stanford University School of Medicine, San Francisco, Calif., and *Edith Green,* Television Cooking Expert, San Francisco, Calif. 94 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1952. Price $3.75.

Connective Tissues, Transactions of the Third Conference, February 14-15, 1952, New York, N. Y., edited by *Charles Ragan,* Department of Medicine, College of Physicians and Surgeons, Columbia University, New York, N. Y. Sponsored by the *Josiah Macy, Jr. Foundation.* 166 pages; illustrated. Printed by Progress Associates, Inc., Caldwell, N. J., 1952. Price $3.50.

Nerve Impulse, Transactions of the Third Conference, March 3 and 4, 1952, New York, N. Y., edited by *H. Houston Merritt,* M. D., Professor of Neurology, College of Physicians and Surgeons, Columbia University, New York, N. Y. Sponsored by the *Josiah Macy, Jr. Foundation.* 176 pages; illustrated. Printed by Progress Associates, Inc., Caldwell, N. J., 1952. Price $3.50.

Prescription for Rebellion, by *Robert Lindner.* 305 pages. Rinehart & Co., Inc., New York, N. Y., publisher, 1952. Price $3.50.

Finality and Form, by *Warren S. McCulloch,* M. D., Professor of Psychiatry and Physiology; Neurophysiologist, Department of Psychiatry, University of Illinois, College of Medicine, Illinois Neuropsychiatric Institute, Chicago, Ill. American Lecture Series, Publication Number 111, A Monograph in American Lectures in Psychology. Edited by *Molly R. Harrower,* Ph. D., Research and Consulting Psychologist, New York, N. Y. 63 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1952. Price $3.75.

The Moral Theory of Behavior, A New Answer to the Enigma of Mental Illness, by *Frank R. Barta,* M. D., F. A. C. P., Director, Department of Psychiatry and Neurology, Creighton University School of Medicine and St. Joseph's Creighton Memorial Hospital, Omaha, Nebraska. American Lecture Series, Publication Number 163, A Monograph in American Lectures in Medicine. 35 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1952. Price $2.

A Manual of Dental Anesthesia, An Illustrated Guide for Student and Practitioner, by *W. Harry Archer,* B. S., M. A., D. D. S., Professor of Oral Surgery and Anesthesia, School of Dentistry, University of Pittsburgh; Head of the Department of Oral Surgery, School of Dentistry; Lecturer, School of Nursing and School of Public Health, University of of Pittsburgh; Chief of the Dental Department of the Magee Hospital; Oral Surgeon and Chief of the Dental Department of the Western Psychiatric Institute and Clinic; Senior Dental Staff Member of the Eye and Ear Hospital; Senior Consultant in Oral Surgery at the Veterans Administration Hospital; Chief of the Dental Department of the Falk Clinic; Diplomate of the American Board of Oral Surgery; Fellow of the American College of Dentists and Fellow of the International College of Anesthetists. 192 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., publisher, 1952.

Correlative Neuroanatomy and Functional Neurology, by *Joseph J. McDonald,* M. S., M. Sc. D., M. D., Professor of Surgery, Columbia University; Attending Surgeon, Presbyterian Hospital, New York; Director of the Surgical Service, Francis Delafield Hospital, New York, and *Joseph G. Chusid,* A. B., M. D., Attending Neurologist, St. Vincent's Hospital, New York. 6th edition. 263 pages; illustrated. Lange Medical Publication. University Medical Publishers, Los Altos, Calif., 1952.

A Manual of Oral Surgery, A Step-by-Step Atlas of Operative Technics, by *W. Harry Archer,* B. S., M. A., D. D. S., Professor of Oral Surgery and Anesthesia, School of Dentistry, University of Pittsburgh; Head of the Department of Oral Surgery, School of Dentistry; Lecturer, School of Nursing and School of Public Health, University of Pittsburgh; Chief of the Dental Department of the Magee Hospital; Oral Surgeon and Chief of the Dental Department of the Western Psychiatric Institute and Clinic; Senior Dental Staff Member of the Eye and Ear Hospital; Senior Consultant in Oral Surgery at the Veterans Administration Hospital; Chief of the Dental Department of the Falk Clinic; Diplomate of the American Board of Oral Surgery; Fellow of the American College of Dentists and Fellow of the International College of Anesthetists. 643 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., publisher, 1952.

The 1952 Year Book of Medicine (May 1951-May 1952), edited by *Paul B. Beeson,* M. D.; *J. Burns Amberson,* M. D.; *William B. Castle,* M. D.; *Tinsley R. Harrison,* M. D.; *George B. Eusterman,* M. D.; and *Robert H. Williams,* M. D. 735 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., publisher, 1952. Price $6.

EFFECT OF ANTIBIOTICS IN ABACTERIAL PNEUMONIA[1]

LeROY HOMER, *First Lieutenant, MC, U. S. A.*
W. N. DONOVAN, *Lieutenant Colonel, MC, U. S. A.*
RYLE A. RADKE, *Colonel, MC, U. S. A.*

MUCH of the pneumonia which is seen in young adults is abacterial in origin and, therefore, is of major concern to the Armed Forces. The experimental and clinical results reported on the efficacy of the various antibiotics in atypical pneumonia are conflicting. It is difficult to evaluate results in animal experiments because the etiologic agent of primary atypical pneumonia has not been definitely isolated. Eaton (2) has demonstrated a definitely beneficial result from the use of aureomycin on the atypical pneumonia which he has transmitted to cotton rats. He was unable to demonstrate any beneficial effects from chloramphenicol.

The reports on the effectiveness of streptomycin (3) and aureomycin (4-15) are largely based on case reports or uncontrolled

(1) U. S. Army Hospital, Ft. Knox, Ky.

(2) Eaton, M. D.: Action of aureomycin and chloromycetin on virus of primary atypical pneumonia. Proc. Soc. Exper. Biol. & Med. 73: 24-29, Jan. 1950.

(3) King, F. P.: Atypical pneumonia treated with streptomycin: a preliminary report on effectiveness of streptomycin in atypical pneumonia. Ann. Int. Med. 34: 141-147, Jan. 1951.

(4) Brainerd, H. D.; Lennette, E. H.; Meiklejohn, C.; Bruyn, H. B., Jr.; and Clark, W. H.: Symposium on antibiotics; clinical evaluation of aureomycin. J. Clin. Investigation 28: 992-1005, Sept. 1949.

(5) Kneeland, Y., Jr.; Rose, H. M.; and Gibson, C. D.: Aureomycin in treatment of primary atypical pneumonia. Am. J. Med. 6: 41-50, Jan. 1949.

(6) Herrell, W. E.: Observations on clinical use of aureomycin. Proc. Staff Meet., Mayo Clin. 24: 612-618, Dec. 7, 1949.

(7) Dowling, H. F.; Lepper, M. H.; Calwell, E. R.; Whelton, R. L.; and Sweet, L. K.: Aureomycin in various infections; report of 180 cases and review of clinical literature. M. Ann. District of Columbia 18: 335-344, July 1949.

(8) Schoenbach, E. B.: Clinical use of aureomycin. M. Ann. District of Columbia 18: 290-293, June 1949.

(9) Olshaker, B.; Ross, S.; Recinos, A., Jr.; and Twible, E.: Aureomycin in treatment of pneumonia in infants and children. New England J. Med. 241: 287-295, Aug. 25, 1949.

(10) Anderson, C. E., Jr.: Treatment of primary atypical pneumonia in children with aureomycin; report of 19 cases. Am. J. Dis. Child. 80: 533-540, Oct. 1950.

(11) Maxwell, F.; Collins, H. S.; and Wells, E. B.: Aureomycin in treatment of primary atypical pneumonia. New England J. Med. 240: 241-246, Feb. 17, 1949.

(12) Schoenbach, E. B., and Bryer, M. S.: Treatment of primary atypical nonbacterial pneumonia with aureomycin. J. A. M. A. 139: 275-280, Jan. 29, 1949.

series. The series reported by Schoenbach et al. *(14)* is difficult to evaluate because it compared a group of patients treated with penicillin and sulfadiazine during the winter of 1946-1947 with another group treated with aureomycin during the winter of 1948-1949. Meiklejohn and Shragg *(16)* and Hilden and Nørregaard *(17)* presented well controlled series affirming the effectiveness of aureomycin. On the other hand, there are several case reports *(7, 14, 18, 19)* and a small controlled series *(20)* in which aureomycin was not found to be of value. Terramycin has been reported to be of the same order of effectiveness as aureomycin in primary atypical pneumonia based on small uncontrolled series *(18, 21, 22)*. Chloramphenicol has also been reported to be of value in this disease *(23, 24)*.

We have attempted to evaluate aureomycin and terramycin in abacterial pneumonia as it occurred at this hospital in the winter of 1951. Because of the overwhelming predominance of abacterial pneumonia at this hospital between January and June 1951, no attempt was made to select patients at the onset of therapy. After the laboratory studies were completed, patients that did not meet our criteria for the diagnosis of abacterial pneumonia were dropped from the series. Our criteria for inclusion in the study group were (1) compatible history and clinical course, (2) relatively normal total leukocyte and differential count, (3) pulmonary infiltrate on roentgenographic examination, (4) absence of pathogens in the sputum, and (5) negative serologic tests for other viral causes of pneumonia.

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(13) Collins, H. S.; Wells, E. B.; Gocke, T. M.; and Finland, M.: Treatment of primary atypical pneumonia with aureomycin. Am. J. Med. 8: 4-20, Jan. 1950.

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(17) Hilden, T., and Nørregaard, S.: Aureomycin in treatment of primary atypical pneumonia (virus pneumonia). Ugesk. laeger 112: 1389-1391, Oct. 5, 1950.

(18) Finland, M.; Gocke, T. M.; Jackson, G. G.; Womack, C. R.; and Kass, E. H.: Clinical studies on terramycin. Ann. New York Acad. Sc. 53: 290-296, Sept. 15, 1950.

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(21) Melcher, G. W., Jr.; Gibson, C. D., Jr.; Rose, H. M.; and Kneeland, Y., Jr.: Terramycin in treatment of pneumococcic and primary atypical pneumonia. J. A. M. A. 143: 1303-1308, Aug. 12, 1950.

(22) Kneeland, Y., Jr., and Melcher, G. W., Jr.: Terramycin in treatment of primary atypical pneumonia. Ann. New York Acad. Sc. 53: 437-439, Sept. 15, 1950.

(23) Wood, E. J.: Atypical pneumonia treated with chloromycetin. Lancet 2: 55-56, July 9, 1949.

(24) Cohen, P. (New York), and Schwartz, R.: Use of chloromycetin in infections; in nonbacterial (atypical undifferentiated respiratory infections). J. Pediat. 37: 23-36, July 1950.

Leukocyte counts were obtained within the first 24 hours of admission and sputum was obtained for culture promptly on admission to the hospital prior to the institution of therapy. Roentgenograms of the chest were obtained at weekly intervals. Blood was drawn for serologic testing at the time of admission and at intervals of 10 days, 3 weeks, and 6 weeks after admission to the hospital. No positive results were obtained for Q fever or the psittacosis-lymphogranuloma venereum group of viruses. Two patients with elevated heterophil agglutination titers were dropped from the study. Many patients were tested for influenza agglutinins but because almost all the patients had been recently immunized with the standard influenza vaccine, these results could not be evaluated and were disregarded. Cold agglutinations and streptococcus MG agglutinations were performed on all patients. Ten significantly elevated titers were found; 5 patients had cold agglutinin titers of 1:32 or over; 3 had a 4-fold or greater increase in their streptococcus MG agglutinations; and 2 had increases in both streptococcus MG and cold agglutinin titers of this order.

The patients admitted in January, February, and March 1951 were designated as group 1. As they were admitted, the first patient was given 200,000 units of crystalline penicillin G intramuscularly every 6 hr. for 20 doses; the second was given 500 mg. of aureomycin by mouth every 4 hr. for 6 doses, then every 6 hr. for from 5 to 10 days; the third was given a placebo-filled capsule (identical in appearance with that of aureomycin) every 4 hr. for 6 doses, then every 6 hr. for about 1 wk.; and so on in rotation.

The patients in group 2 comprised those admitted in April, May, and June 1951. As they were admitted, the first patient was given penicillin as in group 1; the second was given 500 mg. of terramycin by mouth every 6 hr. for 20 doses; the third was given a placebo-filled capsule (identical in appearance with that of terramycin) every 6 hr. for 20 doses; and so on in rotation.

In each group, the patients were quite comparable as regards duration of illness prior to hospitalization, leukocyte counts, and temperature on admission.

RESULTS

Group 1. Eighty-eight percent of those treated with penicillin, 91 percent of those treated with the placebo, and 83 percent of those treated with aureomycin were afebrile at the end of 48 hours of therapy. No patient receiving the placebo was febrile after the third day and no patient receiving aureomycin was febrile after the fourth day. One patient treated with penicillin was febrile 6½ days. Recurrence of a febrile period after being afebrile for over 72 hours occurred in a few patients regardless of therapy (table 1).

TABLE 1. *Duration of temperature elevations (99.5° F. or over)*

Days of treatment	Group 1						Group 2					
	Penicillin		Placebo		Aureomycin		Penicillin		Placebo		Terramycin	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Less than 1	11	46	7	32	8	35	5	29	8	50	9	50
1	6	25	10	45	6	26	5	29	2	13	5	28
2	4	17	3	14	5	22	1	6	1	6	2	11
3	1	4	2	9	3	13	1	6	2	13	1	5.5
4	1	4			1	4	2	12	1	6	1	5.5
5							1	6	1	6		
6	1	4										
7							2	12				
8												
9												
10									1	6		
Total	24		22		23		17		16		18	

TABLE 2. *Duration of pulmonary infiltration on roentgenograms after hospitalization*

Weeks	Group 1						Group 2					
	Penicillin		Placebo		Aureomycin		Penicillin		Placebo		Terramycin	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
1	3	11	3	10	2	6	5	22	6	24	12	43
2	8	28	10	34	10	32	13	57	10	40	12	43
3	8	28	4	14	8	26	3	13	6	24	4	14
4	3	11	4	14	7	23			1	4		
5	2	7	3	10	1	3	2	8	1	4		
6	1	4	1	3								
7	2	7	4	14	2	6			1	4		
Over 7	1	4			1	3						
Total	28		29		31		23		25		28	

After hospitalization, the rate of clearing of the pulmonary infiltration on roentgenograms was closely comparable on all treatment schedules. Sixty-seven percent of those receiving penicillin, 58 percent of those receiving the placebo, and 64 percent of those receiving aureomycin had resolution of their pulmonary infiltrations at the end of 3 weeks. From 85 to 90 percent of all patients had clearing of their pulmonary infiltrations by the end of 6 weeks regardless of therapy. The remainder of the patients took about 2 months for their roentgenograms to return to normal (table 2).

Group 2. Sixty-four percent of patients receiving penicillin, 69 percent of those receiving the placebo, and 89 percent of those receiving terramycin were afebrile after 48 hours. After 4 days of therapy, 82 percent of the penicillin-treated patients, 88 percent of the placebo-treated patients, and all of the terramycin-treated patients were afebrile.

By the end of 2 weeks of hospitalization, 79 percent of those receiving penicillin, 64 percent of those receiving the placebo, and 86 percent of those receiving terramycin had resolution of their pulmonary infiltration on roentgenographic study. At the end of 3 weeks, 92 percent of those receiving penicillin, 88 percent of those treated with a placebo, and all of those treated with terramycin had complete resolution. One patient receiving the placebo had persistence of the pulmonary infiltration after 6 weeks of hospitalization.

Complications were uncommon. Pleural effusions occurred in 2 patients treated with penicillin. In 1 resorption was spontaneous and in the other a diagnostic paracentesis was performed. One patient receiving the placebo developed a pansinusitis and had to be treated with antibiotics.

STATISTICAL ANALYSIS

The results were tested statistically. Those results in which there were less than 30 patients in the smallest group were tested by the chi square method. When the numbers were larger than 30, the results were evaluated by testing for the standard error. There was no significant variation in the rapidity with which the patients in group 1 became afebrile and the rapidity with which their pulmonary infiltrations resolved (table 3). In group 2 there was greater variation. Through the end of the third day there was no significant difference in the rapidity with which the patients became afebrile, but the variation after the fifth day of therapy, when all patients treated with terramycin were afebrile, was significant. Comparison of the rapidity with which the pulmonary infiltrations resolved was of borderline significance.

TABLE 3. *Statistical evaluation of the effect of therapy expressed in chances in 100 that the difference is a chance difference due to sampling*

	Medication					
	Penicillin compared with aureomycin	Placebo compared with aureomycin	Penicillin compared with terramycin	Placebo compared with terramycin	Penicillin compared with aureomycin or terramycin	Placebo compared with aureomycin or terramycin
Effect on the duration of temperature elevation after 5 days of therapy	chance variation	chance variation	1.0	13.0	3.8	7.9
Effect on the rate of resolution of the pulmonary infiltration	52.0*	19.0*	14.7**	1.7**	37.0**	17.0**

*Based on 5 weeks' observation.
**Based on 3 weeks' observation.

Because the patients treated with aureomycin and those treated with terramycin had almost identical responses in their febrile course, the 2 groups were combined and then re-evaluated (table 4). It was definitely demonstrated that aureomycin and terramycin significantly shortened the febrile period in the few patients who would have been febrile beyond the fifth day of hospitalization. In combined series, we could demonstrate no significant effect with any treatment schedule on the rate of pulmonary resolution (table 5).

TABLE 4. *Duration of temperature elevation (over 99.5° F.) in groups 1 and 2 combined*

Days of treatment	Penicillin		Placebo		Aureomycin or terramycin	
	Number	Percent	Number	Percent	Number	Percent
Less than 1	16	39	15	40	17	41
1	11	27	12	31	11	27
2	5	12	4	10	7	17
3	2	5	4	10	4	10
4	3	7	1	3	2	5
5	1	2	1	3		
6	1	2				
7	2	5				
8						
9						
10			1	3		
Total	41		38		41	

DISCUSSION

The numbers of patients in our treatment schedules were small and if those who were mildly ill were excluded, the number of patients whose course was significant becomes even smaller. We believe, however, that we can reach some definite conclusions in view of the fact that we were dealing only with young men who had been in good physical condition prior to their acute illness.

There was no significant variation within the 2 groups when evaluated separately, but when they were combined, it became evident that the broad spectrum antibiotics exerted some effect because no patient receiving these agents was febrile after 5 days of therapy. On the other hand from 6 to 9 percent of those receiving penicillin or a placebo were febrile after this period of time. Neither aureomycin nor terramycin, however, had any significant effect on the rapidity of pulmonary resolution. These pa-

tients who were ill during the warm spring weather had much more rapid clearing of their pulmonary infiltrations than those who were ill during the winter regardless of therapy.

TABLE 5. *Duration of pulmonary infiltration in groups 1 and 2 combined*

Weeks	Penicillin		Placebo		Aureomycin or terramycin	
	Number	Percent	Number	Percent	Number	Percent
1	8	16	9	17	14	24
2	21	40	20	37	22	37
3	11	22	10	19	12	20
4	3	6	5	9	7	12
5	4	8	4	7	1	2
6	1	2	1	2		
7	2	4	5	9	2	3
Over 7	1	2			1	2
Total	51		54		59	

SUMMARY

Abacterial pneumonia, as it commonly occurs, is variable in its course, ranging in severity from an asymptomatic pulmonary infiltration to a severe prostrating illness. It is usually a benign, self-limited disease. Its course is definitely influenced by the season of the year. In view of these facts, it is difficult to evaluate the efficacy of any therapeutic agents. In the series of patients studied here, aureomycin and terramycin had no effect on the rapidity of clearing of the pulmonary infiltration and the effect on their febrile course was not striking. About 90 percent of the patients got well at the same rate of speed with or without antibiotic therapy, but aureomycin and terramycin appeared to prevent the prolonged febrile course that occurred in the remaining 10 percent.

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SMALLPOX

Twenty-one Cases in United Nations Personnel, Korea

SIGURD E. SIVERTSON, *M. D.* (1)

JULIAN B. HYMAN, *Lieutenant, MC, U. S. N.* (2)

FROM October 1950 to April 1951 twenty-one patients with smallpox were seen at a station hospital in Korea.

CLINICAL OBSERVATIONS

The 21 patients varied in age from 4½ months to 54 years. Eighteen of the patients were between 18 and 25 years of age. All were men except for a female dependent, 4½ months old. Thirteen were Americans. Because of the many terminologies confusing the classification of smallpox, the patients were divided into 4 groups: (1) those showing lesions which had run together or coalesced on the face, or on the face and elsewhere on the body, were diagnosed confluent smallpox; (2) those in whom the pox remained individually distinct and the last crust was lost during the third or fourth week of illness were diagnosed discrete smallpox; (3) when the duration of illness was 10 days and the rash easily confused with chickenpox, a diagnosis of varioloid smallpox or alastrim was made; and (4) when bleeding occurred at any stage of the disease a diagnosis of hemorrhagic smallpox was made. The observations are summarized in table 1.

Confluent smallpox. The 8 patients who did not have a smallpox scar stated that "vaccinations never worked" on them. One of the 8 had a vaccinoid reaction of 8 days' duration at the time his smallpox developed. The other 3 were Turks, admitted late in the course of the disease when confluent pox prevented visualization of scars and language barriers made immunization histories unobtainable.

Before the rash developed, chills, fever, headache, backache, nausea, vomiting, and abdominal pain were present for 4 or 5 days in most of these patients, but in 1 they lasted for 8 days. The rash began as a maculopapular eruption often appearing overnight on the face. A prodromal generalized hyperemia was confused with measles and scarlet fever in 1 patient. In another, the

(1) Formerly Chief of Infectious Disease, Station Hospital, Korea; presently a Fellow in Medicine at Mayo Clinic, Rochester, Minn.

(2) Formerly Chief of Laboratories, Station Hospital, Korea; presently stationed at the U. S. Naval Hospital, St. Albans, N. Y.

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TABLE 1. *Clinical data on 21 patients with smallpox*

Group	Number	Childhood scar		History of from 1 to 4 unsuccessful vaccinations in preceding 8 months	Extensive pharyngeal pox	Lymph-adenop-athy	Last crust gone after:
		Absent	Present				
Confluent	11	8*	0	8*	7	11	28-42 days
Discrete	7	4	3	6	0	7	20-28 days
Varioloid	2	0	2	2	0	0	10 days
Hemorrhagic	1	-	-	**	0	1	***
Total	21	12	5	16	7	19	

* History was unobtainable from the other 3 patients.
** History was unobtainable from this patient.
*** Patient died on sixth day; no crusts were present.

papules appeared initially about old psoriatic lesions of the elbows. Seven in this group developed extensive oral and pharyngeal pox, such that mastication and deglutition were not possible. All of these died, whereas, the others who were able to eat and drink lived. Axillary lymphadenopathy was found in every patient, and 2 also had prominent cervical nodes. The nodes were soft, slightly tender, discrete, freely movable, and varied in size from that of a pea to that of a small plum.

Therapy consisted of symptomatic and supportive measures, and antibiotics. One patient was given 1 gram of aureomycin on admission, followed by 500 mg. every 6 hours. Even though no pustular stage was seen, this patient's temperature continued to rise and he died. Another who was given the dosage of aureomycin, plus 300,000 units of procaine penicillin in aluminum monostearate intramuscularly daily survived. No pustular stage was seen in this patient. Two patients were given 1 gram of chloromycetin on admission and 500 mg. every 6 hours plus penicillin; 1 died and 1 lived. Again no pustular stage was seen. Of 4 patients who received penicillin alone, 1 died. The 3 Turks, admitted late in the course of the illness, showed a minimal pustular stage before death, but did not receive penicillin until 2 or 3 days before they died.

Discrete smallpox. Of 7 patients with discrete smallpox, 3 had childhood smallpox scars and subsequent vaccinations had never been successful. The other 4 did not have scars and, although they had been vaccinated at least twice in the preceding 8 months, none were successful. Six of the patients had had fever for 4 or 5 days and the seventh, 8 days before a skin eruption appeared. The rash was typical in onset and progression. Five showed a few oral and pharyngeal pox which did not interfere with swallowing. Axillary lymphadenopathy was found in all of these patients and 2 of them also showed cervical adenopathy. The penicillin schedule previously described was routinely administered and half the adult dose was given to the infant. No pustular stage with rise in temperature occurred. Those started on antibiotic therapy late in the vesicular stage developed a few pustules.

Varioloid smallpox. In both of 2 patients with varioloid smallpox, vaccinations had been unsuccessful since the childhood "take." Pre-eruptive symptoms continued for 5 days in both patients. Neither of them was toxic when admitted and the rash could not be differentiated from that of chickenpox. No mucosal lesions or adenopathy were seen. One patient was diagnosed by culturing the virus from vesicular fluid on the chorio-allantoic membrane of a developing chick embryo. Intradermal inoculation of a rabbit and Paul's test were positive also. The other was

diagnosed clinically by the progression of the rash and particularly by the peripheral blood manifestations. Therapy was no problem and penicillin was given as previously described.

Hemorrhagic smallpox. Although the virus was not cultured from the one patient with hemorrhagic smallpox, the demonstration of Guarnieri's bodies in the skin confirmed the diagnosis. The patient, a 54-year-old sailor, became ill with headache, nausea, vomiting, and abdominal pain about 5 days before admission. Because he was confused and apathetic a detailed history could not be obtained. He was afebrile and had a peculiarly red complexion which had started on his thighs 2 days before admission. The redness was produced by diffuse erythema over his face, arms, trunk, and to a lesser extent on his legs. Generalized petechial, purpuric and ecchymotic lesions were also present, but were most marked in his lower extremities. They seemed to increase in number and intensity during the examination. His liver and spleen were questionably enlarged. He was given a blood transfusion on the evening of admission and bled profusely about the needle. Because of this severe bleeding tendency, all parenteral medications were stopped. Aureomycin was given by mouth. On the following morning, his respirations increased to 40 per minute, rales filled both lung bases, his erythema became more intense, and the purpura progressed and coalesced. He became irrational and cyanotic. Twenty-four hours after admission on the sixth day of his illness, he died. His hemoglobin was 16; his erythrocyte count was 4,210,000; his hematocrit was 49 percent; his leukocyte count was 25,150 with 58 percent lymphocytes, 28 percent neutrophils, 7 percent basophils, 2 percent eosinophils, 2 percent myelocytes, 2 percent blast cells, and 1 percent eosinophilic myelocytes. He had 9 nucleated red blood cells per 100 leukocytes and a platelet count of 33,680. His bleeding time (Duke) was over 1 hour. His sedimentation rate (Wintrobe) was 16. His nonprotein nitrogen was 114 and his creatinine was 5.1. His bone marrow showed a myeloid hyperplasia with a shift to the left. The predominating cells were myelocytes. No foreign cells were seen.

At autopsy, the skin showed diffuse generalized petechias and ecchymoses which were most marked in the lower extremities. Diffuse conjunctival hemorrhages were present bilaterally. The same type of hemorrhagic lesions were seen in small numbers throughout the abdominal subcutaneous tissues, viscera, and retroperitoneal tissues. The spleen weighed 250 grams and its cut surface was beefy red.

Microscopically, the spleen showed marked congestion and great diminution in the size of the lymphoid follicles. The follicle framework remained intact, but most of the lymphocytes had

disappeared. Numerous eosinophils and polymorphonucleocytes were interspersed in the follicular areas. Large numbers of granulocytes including young immature forms and many nucleated red blood cells were seen throughout the pulp and sinusoids. The entire spleen showed evidence of advanced necrosis, manifested by pyknotic nuclei and large numbers of fragmented nuclei.

The general architecture of the lymph nodes was normal, but the follicles were represented by a few small foci and the remainder of the node consisted of framework. Large mononuclear cells with lymphocytelike nuclei and abundant basophilic cytoplasm eosinophils, and granulocytes were scattered throughout the framework and filled the sinusoids. Multiple sections of the skin prepared with Giemsa's stains showed round to oval homogeneous eosinophilic bodies surrounded by a halo in the cytoplasm of the epidermal cells. These were interpreted as Guarnieri's bodies.

Outcome. Eight of the 21 patients (38 percent) died. Four of these, in whom the date of onset could be accurately determined, died on the fourteenth day of the disease.

HEMATOLOGIC OBSERVATIONS

Hematologic studies consisted of periodic hemoglobin determinations, total leukocyte counts, and differential counts. Postmortem bone marrow examinations were made immediately in 5 patients. Most of the patients experienced a drop in hemoglobin to levels as low as 10 grams around the fourteenth day of illness, although some did not experience any significant fall. There was a mild amount of polychromatophilia in the blood of those patients who did have a drop in hemoglobin. Eight patients had nucleated red blood cells during the course of their disease.

Table 2 shows the course of the leukocytic findings in various stages of the disease. During the first 8 days of illness, a leukocytosis due to an increase in neutrophils was noted. This leukocytosis continued from the eighth to the thirteenth day of illness with a relative lymphocytosis producing an equal distribution between lymphocytes and neutrophils. During the critical stage of the disease, about the fourteenth day of illness, when the vesicular stage prevailed, the lymphocytes increased and an absolute lymphocytosis developed. This lymphocytosis and leukocytosis in some patients persisted as late as the thirtieth day of the disease. In others, the leukocytic findings became normal sooner. The patients showing an absolute lymphocytosis or a rising lymphocyte count at the critical stage usually recovered, but those failing to show this or showing a decrease in lymphocytes died. The total leukocyte count ranged from 2,500 to 32,800. Four patients in the series did not have a leukocytosis during the acute phase of the disease.

Qualitative changes occurred in both the myelocytic and lymphocytic series. Twelve of the 21 patients had a shift to the left in the myelocytic series with the presence of myelocytes and frequently younger cells. In all patients the lymphocytes showed changes characterized by extreme variations in size, degree of staining of the nucleus and cytoplasm, and azurophilic granulation of the cytoplasm.

TABLE 2. *Course of leukocytic findings in smallpox*

	1-8 days	9-13 days	14th day or later
Recoveries	Leukocytosis with increased polymorphonuclears	Leukocytosis with slightly increased lymphocytes	Leukocytosis with lymphocytosis
Deaths		Leukocytosis with increased polymorphonuclears	Leukocytosis with increased polymorphonuclears

The bone marrow of the 5 patients studied at autopsy revealed a myeloid hyperplasia with a pronounced shift to the left. The predominant cell was a myelocyte or premyelocyte.

PATHOLOGIC OBSERVATIONS

An autopsy was performed on 4 of those who died. The most revealing observation was a generalized lymphadenopathy. These changes grossly resembled the findings associated with lymphoma. Microscopically, the lymph nodes were hyperplastic in 3 cases and in the fourth had the appearance of lymphadenitis. Guarnieri's bodies of the skin were seen in 3 cases. Surprisingly enough, 2 showed no pneumonia grossly or microscopically. Minimal localized pneumonia was seen in a third. In the fourth, who had had no antimicrobial therapy until 2 days before death, an overwhelming bronchopneumonia was present.

DISCUSSION

All patients admitted to the station hospital were routinely vaccinated against smallpox. Among this group of several thousand, there were a few who, despite repeated daily vaccinations with different lots of vaccine, failed to show a satisfactory response. They did not have a childhood scar and left the impression that there are some persons who may never react to vaccination. In most of these patients, however, a successful take was obtained.

The recognition of smallpox during the prodromal stage and differentiation between the initial rash and that of chickenpox have long been a problem to the clinician. Sometimes it is impossible, except by recovering the virus from vesicular fluid. Certain observations in these cases proved helpful.

In an endemic region, a vaccination history is most important. The disease strikes those who have not had a successful take, despite many vaccinations, or adults who have not had a successful vaccination since a primary vaccination reaction in childhood. Difficulty arose in differentiating a true immune reaction from a foreign protein response *(3)*. Because several patients in this study showed recent immune reactions on their immunization records, the recording of "immune" on an immunization record was never taken at face value. Such a story in a patient with a sustained, toxic, unexplained, febrile illness should immediately place the physician on guard and prompt him to isolate the patient.

The presence of early lymphadenopathy, particularly axillary, lends further weight to the diagnosis of smallpox. Lymphadenopathy in chickenpox is also not uncommon, but in smallpox it seems to appear earlier, i. e., during the pre-eruptive period.

These points, together with the remarkable peripheral blood changes helped make the diagnosis of smallpox in 1 patient before a characteristic rash developed.

Differential criteria between the skin eruption of chickenpox and smallpox can be found in most textbooks. The variability in the developmental stage of the lesions was indeed useful, but the great variation in size of the pox plus their wide erythematous margins proved most valuable in recognizing chickenpox.

Extensive oral, pharyngeal, and laryngeal mucosal lesions were ominous prognostic signs. The patients who were unable to eat or drink because of them, died despite the giving of fluids intravenously and subcutaneously.

A patient was considered noninfectious when the last crust disappeared. The soles of the feet were last to clear. At that time the patient was evacuated. Frequently, when a primary crust was picked off, minute bleeding and extravasation of serum formed a secondary crust, which, to the clinician seeing the case for the first time, led him to believe the patient still infectious. Day-to-day observation for this reason is essential in expediting evacuation.

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(3) Benenson, A. S.: Immediate (so-called "immune") reaction to smallpox vaccination. J. A. M. A. 143: 1238-1240, Aug. 5, 1950.

Hemorrhagic manifestations of smallpox may occur in the pre-eruptive, vesicular, or pustular stage of the disease *(4)*. In the patient herein described, purpuric lesions developed before a typical rash appeared and an erroneous diagnosis of idiopathic thrombocytopenic purpura was made.

The present day hematologic concept holds that in the prodromal stage a leukocytosis due to increased neutrophils exists; in the prepustular eruption, the differential hematologic findings return to normal and a leukopenia may appear; and in the pustular stage, an absolute polymorphonuclear leukocytosis develops. Observations in this study indicate that during the critical prepustular stage, occurring on the fourteenth day of illness, an absolute lymphocytosis occurs in the patients who recover and does not develop in those who die. This provides a valuable prognostic sign *(5)*. Furthermore, qualitative changes in the neutrophils and more particularly the lymphocytes were observed early (in the pre-eruptive stage). This re-emphasizes their usefulness in early diagnosis *(6)*.

In hemorrhagic smallpox, the blood shows a marked granulocytic change, leukocytosis, lymphocytosis, and thrombocytopenia as observed in our patient. It is significant that here is a virus infection, affecting all major blood components much like leukemia *(4)*. With the advent of antibiotics, the course of smallpox has changed. No longer does the green, yellow malodorous pus form in quantities of about 5 liters per day *(7)*. A classical pustular stage with its pyrexia was not seen in any of our patients. Aureomycin, chloramphenicol, and penicillin are all apparently effective in preventing it, but penicillin given intramuscularly is preferred, except in patients with hemorrhagic smallpox, in view of the dysphagia occurring with pharyngeal mucosal lesions.

There was no opportunity for a follow-up in this study. With the elimination of the pustular stage, it seems reasonable to assume that scarring might be greatly reduced.

Dramatic as these changes may be, smallpox is none the less deadly. Despite the apparent elimination of a secondary invader, a mortality rate of nearly 40 percent occurred in this series. The cause of death now is more problematical than when

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bacterial invasion was a factor. The explanation may lie in a profound physiologic disturbance resulting from extensive skin destruction and transudation of fluids and electrolytes.

SUMMARY

Smallpox attacks those who have never had a successful vaccination or who have not had a take since the primary vaccination reaction in childhood. Attention is called to the difficulty in differentiation between a true immune reaction and a foreign protein reaction. The hematologic changes are of diagnostic and prognostic value.

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SITE FOR INTRAMUSCULAR INJECTION

ROBERT W. AUGUSTINE, *Lieutenant Colonel, U. S. A. F. (MC)* (1)
W. E. LANDMESSER, Jr., *Captain, U. S. A. F. (MC)* (1)
MERVEL V. PARKER, *Captain, U. S. A. F. (MC)* (1)
OTIS L. VADEN, *Captain, U. S. A. F. (MC)* (1)

ALTHOUGH the gluteal muscles of the buttock are most often selected for intramuscular injections, this site is not the best available because (1) the area cannot be adequately exposed without turning the bed patient, (2) the buttock of the bed patient is often contaminated by feces, and (3) there is the possibility of damage to underlying nerves and blood vessels. The major objection to the use of the gluteal muscles as the site of choice for intramuscular injection is the possibility of damage to the sciatic nerve. This complication has been reported previously (2, 3), and we know of 3 patients with sciatic palsy resulting from gluteal injections. One recovered and one partially recovered but the third is unimproved.

Sciatic palsy may follow intramuscular injection when the medication is deposited intraneurally or because of careless and negligent insertion of the needle into improper portions of the muscles. The injection is often made into the general region of the hip with little regard for the underlying anatomic structures. The use of the brachium should be avoided because of the relatively small mass of muscle and the danger of injury to the axillary, radial, and ulnar nerves (2).

The best site for intramuscular injections is the lateral aspect of the thigh, the medication being injected into the mass of the vastus lateralis of the quadriceps femoris group (fig. 1). This site is not original with us. Our object is to call attention to it and emphasize its importance. Turner (4) was, as far as we have

(1) U. S. Air Force Hospital, Maxwell Air Force Base, Ala.

(2) Broadbent, T. R.; Odom, G. L.; and Woodhall, B.: Peripheral nerve injuries from administration of penicillin; report of 4 clinical cases. J. A. M. A. 140: 1008-1010, July 23, 1949.

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(4) Turner, G. G.: Site for intramuscular injections. (Letter to Editor of the Lancet) Lancet 199: 819, Oct. 16, 1920.

been able to ascertain, the first to advocate the use of the vastus lateralis. This site was subsequently recommended in response to a query addressed to the editor of the *British Medical Journal* (5); stressed again by Turner (6) in 1944 and recently by Krishna (7). These authors have unanimously favored the lateral aspect of the thigh over the gluteal region.

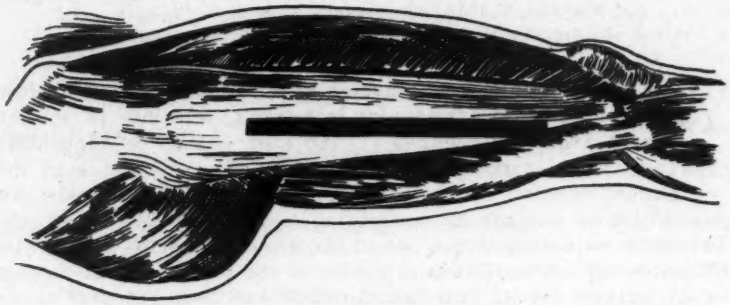

Figure 1. Lateral view of fascia and muscle mass of thigh showing favored site (blacked in) for intramuscular injection. (Adapted from Manual of Surgical Anatomy, Medical Departments, U. S. Army and U. S. Navy, 1918.)

Physicians in this country have neglected the advantages of using this site for intramuscular therapy. We are not familiar with any reference to it in American medical literature. The recommended area is readily available in either the prone or supine patient; is large enough to permit repeated injections; and contains neither major nerves nor vessels (fig. 2).

The injections are made into the muscles of the thigh along a line extending from the greater trochanter above to the lateral femoral condyle below. Limiting this line by a handsbreadth above the knee and the same distance below the greater trochanter gives ample space for multiple injections placed 1 inch apart. With the patient sitting or lying supine, the needle is inserted parallel with the floor or directed slightly toward the anterior aspect of the thigh (fig. 3). A fast harpoon thrust will sink the needle to the hub painlessly. For adults, a 1½-inch needle is used. Should the needle strike the femur, pain or tissue injury will be negligible. Aspiration must be attempted prior to injection to avoid intravascular deposition of the medication.

(5) Anonymous: Site for intramuscular injection. (Letters, Notes, and Answers) Brit. M. J. 1: 775, June 19, 1943.

(6) Turner, G. G.: Brit. M. J. July 8, 1944. Cited in footnote reference (7).

(7) Krishna, G.: Intramuscular therapy. Antiseptic 45: 703-710, Oct. 1948.

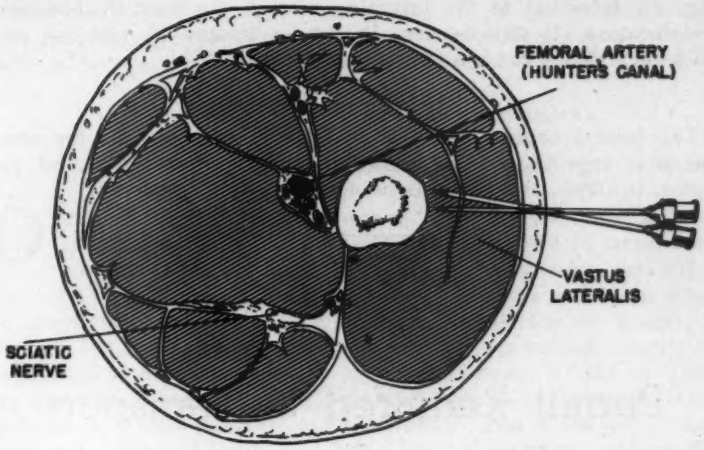

Figure 2. Cross section of middle third of the thigh showing muscle mass, vital structures, and optimum positions of needle. (Adapted from Manual of Surgical Anatomy, Medical Departments, U. S. Army and U. S. Navy, 1918.)

In children, because penetration to the sciatic nerve or great vessels is possible, a shorter needle should be used.

Hunter's canal, lying in the medial thigh, is not reached unless the injection is made very carelessly and too far posteriorly

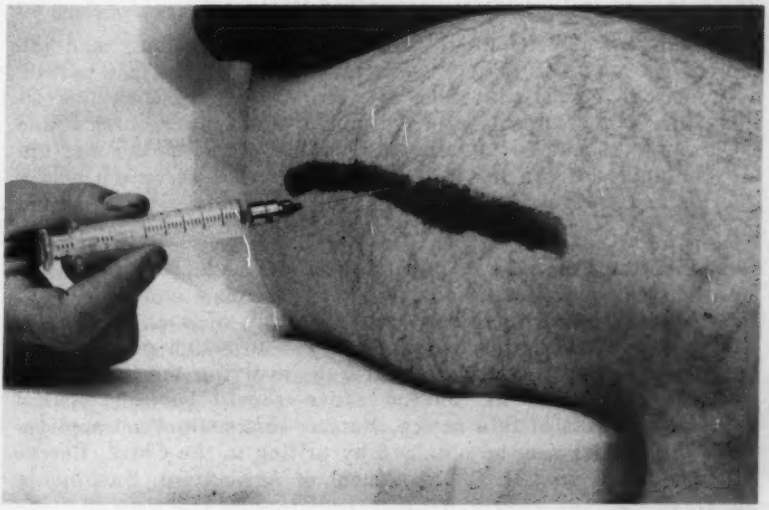

Figure 3. Oblique view of external aspect of thigh showing angle of syringe for injection. Black line indicates area which may be used for injections.

(fig. 2). Infection at the injection site is the most troublesome complication. If this occurs, it can be treated by incision and drainage without damage to vital structures.

CONCLUSIONS

The lateral aspect of the thigh is the site of choice for intramuscular injections. The area is easily accessible in all patients, is large, and contains no major vessels or nerves.

Permit Required to Transport Disease Organisms

In recent months several instances of illegal movement of animal disease organisms and vectors in interstate commerce have come to the attention of the U. S. Department of Agriculture. Conditions under which restricted organisms and vectors can be moved under permit are explained in the Department's Bureau of Animal Industry Order 381, Part 122, entitled "Rules and Regulations Relating to Viruses, Serums, Toxins, and Analogous Products, and to Certain Organisms and Vectors."

Because of the inherent danger of such movements and the increasing need for taking every precaution against the spread of infectious animal diseases, all laboratories, research institutions, and others dealing with animal disease organisms and vectors are requested to comply with this order. Movements are allowed under permit only when such shipments serve the public interest and after ample safeguards are provided to protect against the further dissemination of such agents.

Application for such a permit shall be made *in advance of shipment* and each permit shall specify the name and address of the consignee, the true name and character of each of the organisms or vectors involved, and the use to which each will be put. All persons engaged in research involving the use of organisms or vectors as defined above should become familiar with the contents of this notice. Further information and applications for permits may be obtained by writing to the Chief, Bureau of Animal Industry, U. S. Department of Agriculture, Washington 25, D. C.

FUSOSPIROCHETAL STOMATITIS

Treatment With Aureomycin and Terramycin

EUGENE T. BORISH, *Lieutenant, DC, U. S. N.* (1)

ORAL fusospirochetal infections (Vincent's infection or trench mouth, et cetera) present a variety of acute and chronic clinical forms (2). These conditions are often confused with the more diffuse types of gingival infection such as streptococcal and staphylococcal stomatitis or gingivitis caused by the presence of calculus. The lessened degree of tissue resistance attributed to local interference of the nutrition due to subgingival calculus may predispose to invasion by large numbers of *Fusobacterium plauti-vincenti*. The disease is most common in young adults. Causative factors may include insufficient rest, emotional stress, unbalanced or inadequate nutrition, consumption of alcoholic beverages, and direct infection by contact. Poor oral hygiene frequently contributes to the incidence of this disease but is not necessarily an etiologic factor. Studies of the prevalence of fusospirochetal stomatitis among servicemen aboard ship are particularly interesting because of their social habits, age, physical status, and generally good oral hygiene. Oral examinations conducted on 450 enlisted men aboard this ship disclosed a few patients with fusospirochetal stomatitis. Of the men examined, 11 exhibited sufficient subjective and objective clinical symptoms to warrant laboratory study. Six of these cases were of a chronic nature and 5 patients presented acute symptoms. A positive diagnosis was made in all 11 patients by using bacteriologic smears and, in a few, by dark-field microscopic examination. Clinically, the 5 patients presented the characteristic punched-out ulcerations of the interdental papillas, pain in these areas, a gray exudate or slough in the interdental crevices, a metallic taste (present in 2), profuse local hemorrhage on pressure, and a feeling of "soreness" in the teeth. The infected areas were mostly confined to 2 or 3 teeth but in one particularly acute case the entire left mandibular and maxillary dentition was involved. This patient had a temperature of 101.6° F. and a slight increase in the leukocyte count.

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(1) U. S. S. *Mindoro*.

(2) Burket, L. W.: Oral Medicine: Diagnosis, Treatment. J. B. Lippincott Co., Philadelphia, Pa., 1946.

Dark-field microscopic examination revealed large numbers of *Borrelia vincenti*, lesser numbers of *F. plauti-vincenti*, and what appeared to be scattered diplococci. A stained smear revealed these organisms and large clusters of pus cells.

TREATMENT

Topical application has been employed by Rothner et al. (3), using penicillin, and by Goldman and Bloom (4), using aureomycin. I used a mixture of aureomycin and terramycin in a large number of patients prior to its use on those herein reported with gratifying results. The lesions are forcibly sprayed with a three-quarter strength solution of hydrogen peroxide, dried gently by means of the air blast, and isolated by blocking with cotton rolls. A paste of fairly heavy consistency is made by mixing 50 mg. of aureomycin and 250 mg. of terramycin with several drops of distilled sterile water. This is applied generously to the lesions with a broad-bladed plastic instrument, care being taken to force the paste into the crevices surrounding the necks of the teeth and the interdental spaces. The paste is allowed to remain in place for at least 20 minutes. At the expiration of this time the cotton rolls are removed and the patient allowed to flush the mouth with ordinary tap water. He is instructed to avoid transmitting the disease to others, to discard the present toothbrush, to take a bland diet, and to return to the dental clinic on the following morning for further treatment.

The 5 patients with fusospirochetal stomatitis concerned in this report returned on the following day with a complete remission of subjective symptoms. The gray slough had almost completely disappeared and the angry appearance of the tissues had lessened. The local hemorrhagic tendencies persisted. Improvement was so dramatic, however, that the removal of secondary causes could be undertaken immediately. Bacteriologic smears revealed a marked decrease in the number of causative organisms. Follow-up treatments consisted of continued applications of the aureomycin-terramycin pack, elimination of calculus and overhanging restorations, and instruction in proper tooth brushing technic.

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(3) Rothner, J. T.; Cobe, H. M.; Rosenthal, S. L.; and Bailin, J.: Adhesive penicillin ointment for topical application. J. Dent. Research 28: 544-548, Dec. 1949.

(4) Goldman, H. M., and Bloom, J.: Topical application of aureomycin for treatment of acute phase of ulcerative necrotizing gingivitis (Vincent's infection). Oral Surg. 3: 1148-1150, Sept. 1950.

CONCLUSIONS

The therapeutic value of an aureomycin-terramycin mixture is extremely efficacious in the treatment of fusospirochetal stomatitis. It is easy to apply. The only disadvantage is the high cost of these drugs.

ADDENDUM: Excellent results have also been obtained by using the aureomycin-terramycin mixture incorporated with butyn sulfate-metaphen ointment and iodoform gauze in the treatment of the so-called dry socket.

BOOKS RECEIVED

1952 Year Book of Obstetrics and Gynecology (July 1951-June 1952), edited by *J. P. Greenhill,* B. S., M. D., F. A. C. S., Professor of Gynecology, Cook County Graduate School of Medicine; Attending Gynecologist, Cook County Hospital; Attending Obstetrician and Gynecologist, Michael Reese Hospital; Associate Staff, Chicago Lying-in Hospital; Author of Office Gynecology, Obstetrics in General Practice and Principles and Practice of Obstetrics. 575 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1952. Price $5.50.

Synopsis of Pathology, by *W. A. D. Anderson,* M. A., M. D., F. A. C. P., Professor of Pathology, Marquette University School of Medicine; Pathologist, St. Joseph's Hospital, Milwaukee, Wis. 3d edition. 788 pages; illustrated. The C. V. Mosby Co., St. Louis, Mo., publisher, 1952. Price $8.

The Mechanisms of Disease, A Study of the Autonomic Nervous System, the Endocrine System, and the Electrolytes in their Relationship to Clinical Medicine, by *Joseph Stambul,* M. D., Chief of the Cardiovascular Department of Albert Einstein Medical Center (Eastern Division); Cardiologist to Deborah Sanatorium. Foreword by *George Morris Piersol,* B. S., M. D., Professor of Medicine, Graduate School of Medicine, University of Pennsylvania; Editor-in-Chief, The Cyclopedia of Medicine. 746 pages. Froben Press, Inc., New York, N. Y., publisher, 1952. Price $15.

United States Army in World War II, Pictorial Record, The War Against Japan. 471 pages; illustrated. *Office of the Chief of Military History, Department of the Army,* Washington, D. C., 1952. Printed by U. S. Government Printing Office, Washington, D. C., 1952.

Forensic Psychiatry, by *Henry A. Davidson,* M. D., American Academy of Sciences; Fellow, American Psychiatric Association. 398 pages. Ronald Press Co., New York, N. Y., publisher, 1952. Price $8.

Infrared Photography in Medicine, by *Leo C. Massopust,* Sr., Director, Department of Art and Photography, Marquette University School of Medicine, Milwaukee, Wis. 53 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1952. Price $2.75.

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Pharmacology and Therapeutics, by *Charles Solomon,* M. D., F. A. C. P., Lecturer in Medicine, State University, College of Medicine, New York City; Attending Physician, Jewish Hospital of Brooklyn; Attending Physician, Jewish Sanitarium and Hospital for Chronic Diseases; Lecturer in Pharmacology and Therapeutics, School of Nursing, Jewish Hospital; Director of Graduate Course in Applied Pharmacology and Therapeutics, Brooklyn College of Pharmacy; with the collaboration of *Elizabeth S. Gill,* B. S., R. N., Instructor in Nursing, Department of Nursing, Faculty of Medicine, Columbia University, Presbyterian Hospital School of Nursing. 6th edition. 656 pages; illustrated. J. B. Lippincott Co., Philadelphia, Pa., publishers, 1952.

Ophthalmic Plastic Surgery, by *Sidney A. Fox,* M. S. (Ophth.), M. D., F. A. C. S., Assistant Clinical Professor of Ophthalmology, New York University Post-Graduate Medical School; Attending Ophthalmologist, Goldwater Memorial and Bronx Veterans Administration Hospitals; Associate Ophthalmologist, Hospital for Joint Diseases; Assistant Surgeon Ophthalmologist, Bellevue Hospital, New York. 290 pages; illustrated. Grune & Stratton, Inc., New York, N. Y., publishers, 1952. Price $15.

Physiological Bases of Gynecology and Obstetrics, by *S. R. M. Reynolds,* M. A., Ph. D., D. Sc., Dr. honoris causa, Staff Member, Physiologist, Department of Embryology, Carnegie Institution of Washington; Lecturer in Obstetrics, The Johns Hopkins Medical School, Baltimore, Md.; Profesor honoris causa, Facultad de Medicina, Universidad de la Republica Oriental del Uruguay, Montevideo, Uruguay. 153 pages. Charles C Thomas, Publisher, Springfield, Ill., 1952. Price $5.50.

Handbook of Tropical Dermatology and Medical Mycology, edited by *R. D. G. Ph. Simons,* Amsterdam, Senior Lecturer at the Dermatological Clinic of the University of Leyden; Dermatologist in Charge at the Civilian Hospital, Amsterdam. Volume I. 845 pages; illustrated. Elsevier Publishing Co., New York, N. Y., publishers, 1952. Price $15.

The Newer Knowledge of Hygiene in Diet, by *J. Sim Wallace,* M. D., C. M., D. SC., L. D. S., F. D. S., R. C. S., Eng., F. A. C. D., Vice-President of the Food Education Society; Hon. Fellow Society of M. O. H.; (Neech Prize, 1928-29); Hon. Member B. D. A.; Past President of the Brit. Soc. for the Study of Orthodontics; Emeritus Lecturer in Preventive Dentistry, King's College Hospital; Formerly Dental Surgeon and Lecturer on Dental Surgery and Pathology, London Hospital; John Tomes Prize (1918-21) and Cartwright Medal and Prize (1920-25) R. C. S. Eng. 264 pages. Dental Items of Interest Publishing Co., Inc., Brooklyn, N. Y., publishers, 1952.

Rheumatic Diseases, Diagnosis and Treatment, by *Eugene F. Traut,* M. D., F. A. C. P., Associate (Rush) Clinical Professor of Medicine, University of Illinois; Attending Physician to the Cook County Hospital and to the West Suburban Hospital, Oak Park, Ill.; Associate Attending Physician to the Presbyterian Hospital of Chicago; Director of the Arthritis Clinic of Cook County Hospital; Lecturer on Arthritis in the Cook County Graduate School of Medicine; Member of the American Rheumatism Association. 942 pages; 192 illustrations. The C. V. Mosby Co., St. Louis, Mo., publisher, 1952.

Textbook of Surgery, edited by *H. F. Moseley,* M. A., D. M., M. Ch. (Oxon), F. A. C. S., F. R. C. S. (Eng.), F. R. C. S. (C), Assistant Professor of Surgery, McGill University; Associate Surgeon, Royal Victoria Hospital, Montreal, Canada. With foreword by *G. Gavin Miller,* M. D., C. M., M. Sc., F. R. C. S. (C), F. A. C. S., Chairman of the Surgical Department, McGill University; Surgeon-in-Chief, Royal Victoria Hospital, Montreal, Canada. 896 pages; 460 text illustrations and 46 color plates. The C. V. Mosby Co., St. Louis, Mo., publisher, 1952. Price $15.

LEADERSHIP AND THE INCIDENCE OF TRENCH FOOT

EUGENE R. INWOOD, *Colonel, MC, U. S. A. (1)*

PHYSICIANS new to the Army do not always appreciate the amount of assistance a properly oriented and enthusiastic commander can give them nor do they always see why it is the commanding officer's duty to make decisions on medical matters. A medical officer, however, receives his authority from the commander and acts for him in giving commands to nonmedical personnel *(2)*. Actually, he is acting in a staff capacity as medical adviser and in much the same way as does the physician in civil life *(3)*. In an epidemic of diphtheria, for instance, a civilian physician may see a real need to immunize all the children in his particular neighborhood so that his own children may continue their normal activities without undue risk, but he cannot carry out such a program unless each parent consents.

In the Army there is a close parallel. It is the commander who, subject to the regulations given him by the higher commanders and on the recommendations of his medical adviser, makes the medical decisions for all of his men *(4)*. The Army physician, however, has other resources on which he may depend when he is unable to convince a commander of the need for his recommendations. The regulations may support him and also assist the commander in arriving at a decision. He may seek consultation with other medical officers and learn of new approaches, but he is not free to withdraw from the case. The medical officer may report a situation to the next larger unit surgeon who in turn may take such action as he considers necessary. This action may consist of an informal visit, with verbal suggestions being made, or it may be a formal inspection with a written recommendation to his own commander to exert pressure in carrying out the first medical officer's recommendations. In some instances the first medical officer, in his frustration and anxiety, may be unduly disturbed about a minor situation, or he may have forgotten some very valid argument which would be convincing.

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(1) Neuropsychiatric Service, Walter Reed Army Hospital, Washington, D. C.

(2) Army Regulations, 600-20, paragraph 3c, 5 May 1950.

(3) Army Regulations, 40-10, paragraph 4b, 8 Feb. 1952.

(4) Saving lives on the battlefield. Officer's Call 4: No. 4, p. 10, 1952.

Whether in the Army or in civil life, the physician does not have absolute authority. It is important, therefore, to consider the relationship between the medical officer and the commanding officer with respect to compulsory treatment and a program which experience has shown depends not only on the interest and enthusiasm of the medical officer and his staff, but also on the support and active co-operation of the commander. With good leadership and support, extensive medical programs can be carried out and the health of the troops protected. With poor leadership, the end result, regardless of the efforts of the physician and his staff, cannot be satisfactory.

The unit commander can be a bulwark of support in assisting the physician. It has been fully demonstrated that some things such as typhoid vaccination, malarial prophylaxis, and other preventive measures cannot be controlled without compulsory treatment, but there are many conditions which cannot be controlled by immunization and in which the prophylactic treatment is more involved. Such a condition is trench foot. In view of this fact it becomes pertinent to review the relation of leadership to the incidence of trench foot in combat units. This relationship is borne out by an incident which occurred in a combat division in the European Theater of Operations in the late winter of 1944 and the early spring of 1945. This experience shows how in one infantry division when the incidence of trench foot became abnormally high, the recommendations of the division surgeon were completely approved by the commander, and how in 2 regiments of the division the condition was successfully controlled, and in the third regiment was only partially controlled in spite of the efforts of the medical officers to carry out prophylactic treatment as directed.

In the fall of 1944, the division was alerted for overseas movement. The unit had been training for more than a year, but progress through the unit training stage had never been accomplished because individual replacements in all grades were constantly being transferred overseas. A short time before entering the staging area, a series of training inspections was carried out. During these inspections it was discovered that some of the new men were completely unaware of the difference between trench foot and athlete's foot. A series of pictures on trench foot in all its stages, with emphasis upon early detection and treatment, was shown to the entire command. When the unit left for overseas, every man knew what trench foot was and that it was a dangerous condition which, if neglected, could mean the loss of a foot. A few weeks later, however, when the 3 infantry regiments were in combat in France, with the temperature several degrees below freezing, men began reporting back with suspected trench foot. It became apparent then that overemphasis had been

placed on the early detection of the condition and that the earlier training which had dealt with prophylaxis had been forgotten.

On 14 January 1945, at a conference between the division surgeon and the corps surgeon, it was determined that something had to be done to control trench foot After 22 days of combat, one regiment had had 41 cases; another, 172; and the third, 84. The methods of prophylaxis were reviewed and the following recommendations made:

1. All men who were constantly exposed to freezing weather were to take their shoes and socks off once each day and rub their feet for 10 minutes.

2. Each soldier was to be provided with an extra pair of socks to carry on his person between his undershirt and his O. D. shirt so that he would have available 1 dry pair for a daily change. An effort was to be made to send up clean socks each day with the rations, but if this became impossible each man would still have the extra pair of dried socks which he carried on him for emergency wear.

3. Unit leaders were to supervise such procedures and were to set an example by themselves complying.

4. Unit surgeons were to inspect the men being sent to the rear more carefully, evacuating only those who were definitely incapacitated. They were to visit all units down to platoons and give necessary instructions and advice. These visits were to be made as frequently as possible—preferably daily.

The plan was approved by the division commander and regimental commanders were asked to come to the command post that same day. The G4, present at the meeting, agreed to arrange for laundry service and to send clean socks along with the rations. The commanding general issued explicit instructions endorsing the plan and stressing the need for such a program. He emphasized the fact that the unit had to conserve the men it had, that replacements were in short supply and often were completely inexperienced.

Two of the regimental commanders were enthusiastic in their support. Following the general's remarks, the third regimental commander said that: (1) it was a good but impractical idea; (2) his men, only the day before, had captured 2 Germans while in the act of rubbing their feet; (3) his men were too busy looking for Germans to take the necessary time out; (4) his men would not carry wet socks next to them until they dried; they would not put on dirty socks; and he doubted if there were any clean ones available to be sent up with the rations.

These objections were met with the following explanations: Men in foxholes would cover each other while taking turns rubbing their feet. Thus they would not be caught the way his men had caught the German soldiers. Men in other units were carrying their socks in the directed manner and were not objecting to it. The general further pointed out that there were bound to be periods at some time in each day when even the busiest men would have time to carry out this method of prophylaxis if the unit leaders worked on the plan and set a good example themselves. The reluctant commander agreed to try it, but it was evident that he still had some misgivings.

TABLE 1. *Number of patients with trench foot in 3 regiments*

1945	Regiment 1	Regiment 2	Regiment 3
Prior to 15 January	41	172	84
16-31 January	3	3	17
February	6	16	78
March	12	8	46
Total	62	199	225
Number of new cases after instituting control measures	21	27	141

Following the inauguration of this plan, there was an immediate drop in the incidence of trench foot (table 1). The program of prophylaxis was wholeheartedly supported by medical officers throughout the division. Inspections, down to the platoon level, were made daily by unit surgeons or their assistants. The commanders of the first and second regiments were enthusiastic about the project but the commander of the third regiment believed it could not be effective. In all units the medical officers made a sincere effort to carry out the program. All 3 regiments were engaged in the same type of defensive combat, in the same type of terrain, and under the same weather conditions.

In February, the regiments continued in the same tactical situation. On 17 February a limited attack was made by all 3 regiments. The weather was cold, the terrain hilly, and the ground was covered with a varying amount of snow. Again the first and second regiments had only a few cases as compared with the third regiment whose commander had completely lost faith in the plan (table 1). He believed that the attack was the important thing and that his men were too busy to care for their feet. He did not continually emphasize the matter as did the other commanders. The medical officers in all 3 units were

unanimous in their support and showed about the same degree of diligence in their part of the program.

In March, similar results were noted (table 1). The weather was warmer and the temperature above freezing much of the time, but this was offset by certain other factors. The first and third regiments had been attacking for several days across a flat area where the foxholes had a tendency to fill with water. At night the water froze. It was impossible for the men to keep themselves dry if they had to remain in foxholes. The second regiment was on higher ground where the foxholes did not fill with water. No new cases were reported after March. The comparative results in these 3 regiments of the same size and operating under similar circumstances reflected the attitudes of the regimental commanders and showed how effective a control program can be with command support. In some situations it is difficult to obtain the full support of the commander and such a unit will be less successful in maintaining the health of the troops than when the commander offers his full support. The unit surgeon can do much to assist in developing this co-operation.

During the training period, while a combat unit is being developed, the surgeon has an opportunity to establish himself within the unit in a position of security and confidence. He can demonstrate to the commander that his medical opinions are sound and reliable and that he has a thorough understanding of his job. If there exists between commander and surgeon a feeling of mutual dependability, the commander may feel free in allowing the surgeon to do his work and may accept his medical recommendations and programs with confidence. On the other hand, if the commander does not have confidence in the ability of the surgeon, he will be more likely to question the judgment of the surgeon. What is true of trench foot is also true of other medical conditions.

BOOK REVIEWS

Claude Bernard and the Experimental Method in Medicine, by *J. M. D. Olmsted*, Professor of Physiology at the University of California; Membre Correspondant de la Société Philomathique de Paris. Prix Binoux, Académie des Sciences, 1949, and *E. Harris Olmsted*. 277 pages. Henry Schuman, Inc., New York, N. Y., publisher, 1952. Price $4.

This latest volume in the publisher's Life of Science Library is a biography of the pioneer in experimental medicine in the nineteenth century. A previous one was devoted to John Hunter.

It is of interest that both were poor country boys who, the one in London, the other in Paris, carved out great careers and erected landmarks in the history of medicine.

As a young medical student, Bernard came under the influence of the physiologist, Magendie, became his assistant, and eventually succeeded him as Professor of Medicine at the College of France. His entire life was devoted to research and teaching.

Bernard's fame rests principally on three discoveries and the demonstration of the experimental method in arriving at his results. The three great advances in physiology we owe to Bernard are the demonstration of the glycogenic function of the liver, the pancreatic digestion, and the vasomotor mechanism. Much of modern gastroenterology and cardiology rests on these discoveries, each of which would be sufficient for a Nobel Prize award today. The authors of this biography (incidentally a husband and wife team) have given a fine account of Bernard's life and work. They give an interesting picture of the man himself, from his first appearance in Paris as an awkward young countryman to his position as a famous professor with the ribbon of the Legion of Honor in his buttonhole. They mention Bernard's reaction to women medical and science students, then a good deal of a novelty in academic circles. He speaks of them as "Doctoresses" and says that one of them but 17 or 18 "has a heart of steel." Altogether it is an excellent and satisfying book. This reviewer would like to have seen more illustrations though there is a fine photograph of Bernard and a very attractive book jacket.

—Capt. L. H. Roddis, MC, U. S. N. (Ret.)

Sterility, Its Cause and Its Treatment, by *J. Jay Rommer,* A. B., Ph. D., M. D., A. I. C. S., Gynecological Staff, Beth Israel Hospital, Newark, N. J.; Member: American Genetics Association, American Society for the Study of Sterility, New Jersey Neuropsychiatric Association; Fellow, New Jersey Obstetric and Gynecological Society. Part I: The Infertile Female. Part II: The Infertile Male. 424 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1952. Price $12.50.

The author has accomplished his purpose in this book, namely, to provide a reference book touching on every conceivable aspect of human sterility. Each problem is discussed briefly, but an unusually extensive bibliography is given. Dr. Rommer emphasizes the fact that many problems connected with the treatment of sterility remain unsolved.

—Commander H. J. Hunter, MC, U. S. N.

TECHNIC OF INTRA-ARTERIAL TRANSFUSION[1]

SAM F. SEELEY, *Brigadier General, MC, U. S. A.*
RUSSELL M. NELSON, *First Lieutenant, MC, U. S. A. R.*
SIGMUND A. WESOLOWSKI, *First Lieutenant, MC, U. S. A. R.*

THE management of the patient in hemorrhagic shock depends on prompt correction of the etiologic factors. Transfusion of whole blood is the treatment of choice to combat the effects of hemorrhage. Ordinarily, it is possible to replace the volume of lost blood by intravenous administration. In instances of profound shock, especially when the status of the myocardium may be in jeopardy, transfusion by the arterial route has been practiced widely. The medical literature pertaining to the evolution of and experience with intra-arterial transfusion has been reviewed recently (2), and the purpose of this article is to report on the technics involved in the administration of blood by artery.

The basic considerations are (1) the condition of the patient, (2) the selection and manipulation of the artery, (3) the choice of needle, (4) the type of pressure apparatus, (5) the rate and volume of blood required, and (6) the hazards associated with arterial transfusion.

THE CONDITION OF THE PATIENT

The condition of the patient in deep shock must be accurately evaluated and derangements of physiology corrected. Especially in the wounded, factors in addition to hemorrhage may participate in causing shock, such as tension pneumothorax, pulmonary embolus, pericardial tamponade, sepsis, decreased peripheral resistance, fat embolism, and dehydration. The patient who fails to respond as expected should be assessed for any of these complications as well as for continued occult bleeding.

THE SELECTION AND MANIPULATION OF THE ARTERY

Because there are no valves within the arteries, distribution throughout the arterial system will not depend on which artery is used. There are many reports of use of the thoracic and abdominal aorta for arterial transfusion during surgical operations.

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(1) From the Division of Surgery, Army Medical Service Graduate School, Walter Reed Army Medical Center, Washington, D. C.

(2) Seeley, S. F., and Nelson, R. M.: Intra-arterial transfusion. Surg., Gynec. & Obst. 94: suppl. 209-214, Mar. 1952.

By virtue of relative accessibility, either the femoral artery or the radial artery is most commonly used. Although a percutaneous insertion of the needle is often successful, surgical exposure of the artery is recommended, especially when the state of shock is so profound that peripheral arterial pulsations are not detectable.

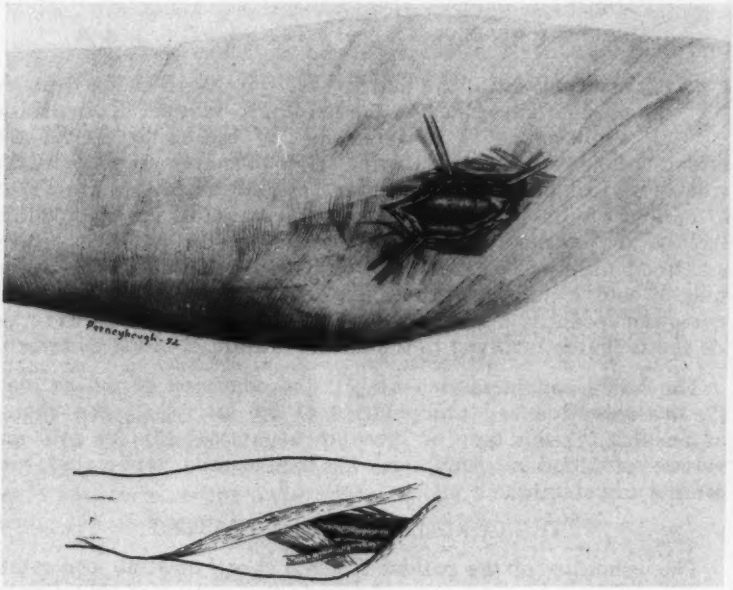

Figure 1. The femoral artery is exposed between the nerve and the vein. The femoral sheath has been incised. The right femoral triangle is shown diagrammatically below. Note the relationship of the great saphenous vein. Its tributaries are not shown. (Courtesy of Dr. Othmar Solnitzky.)

In order to expose the femoral artery, a vertical incision may be made over the femoral triangle, which is bounded by the inguinal ligament, sartorius muscle, and adductor longus muscle (fig. 1). It is enclosed in the femoral sheath, and lies lateral to the vein. (A mnemonic aid to recall the relationship of these structures is "NAVEL" for Nerve, Artery, Vein, Empty space, and Lacunar ligament, reading from lateral to medial.) Tributaries of the great saphenous vein often are encountered in the dissection, and may be ligated.

The radial artery may be exposed just medial to the styloid process of the radius. Figure 2 shows the anatomic relations. For the sake of illustration, the artist has depicted a longitudinal incision, but in practice, it is preferable to make an incision in a line parallel with the flexion creases at the wrist.

A strand of suture material may be placed under the exposed vessel for control, but it should not be tied about the artery. If spasm should occur, the local injection of procaine solution or the application of papaverine packs may be used.

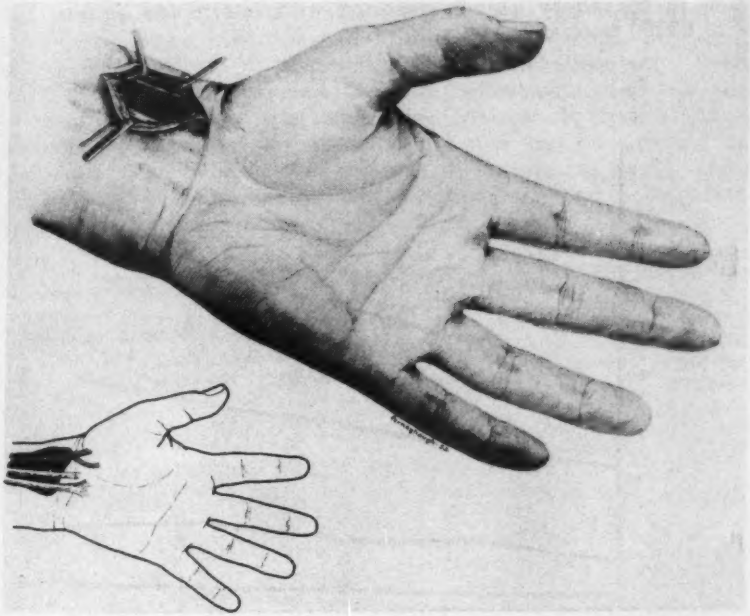

Figure 2. Anatomic relationship of the radial artery at the wrist. The relation of the artery to the styloid of the radius and the tendons of the flexor carpi radialis and palmaris longus muscles is shown diagrammatically below. (Courtesy of Dr. Othmar Solnitzky.)

THE NEEDLE

Because a rapid rate of blood flow is desired, a large needle must be used. Figure 3 shows the comparative rates of flow of blood under controlled pressure through various-sized needles. It may be seen that if 200 mm. Hg. pressure is applied to the blood, and if a flow of at least 200 ml. per minute is desired, a 15-gage needle is required. The entire shaft of the needle is threaded into the artery, and it may be maintained in place by transfixing a suture tied about the hub of the needle to adjacent subcutaneous tissue. The artery will be damaged if the needle is secured by means of a ligature about the artery. The incidence of bleeding after withdrawal of the needle is minimized by (a) having initially inserted the needle so that the cutting edge enters the artery

vertically rather than horizontally and (b) by maintaining digital compression over the puncture site for a full 10-minute period. The artery should not be ligated because gangrene of the distal member will follow in certain instances if this is routinely done. If bleeding persists after the period of compression, the perforation in the artery may be repaired with interrupted sutures of No. 00000 arterial or eye silk.

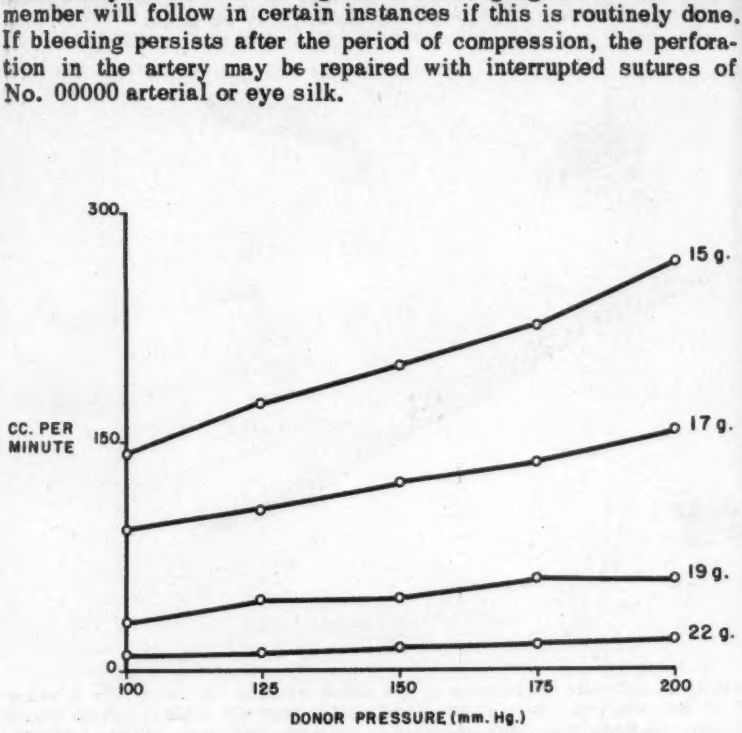

Figure 3. Graph showing the rate of flow of blood through various-sized needles if an artificial resistance of 60 mm. Hg. is applied to the system.

THE APPARATUS

Most hospitals have developed standard means for transfusion of blood under pressure, especially where many patients in shock are treated. There are, however, other instances in which a physician's desire to administer blood by artery is needlessly quelled because of the lack of a specialized pressure apparatus. Such an apparatus is not essential. All that is needed is some means by which the pressure in the transfusion flask can be elevated above that of the blood in the artery so that the direction of flow will be from the blood bottle into the artery. The simplest and most practical device for accomplishing this is a sphygmomanometer bulb, connected by tubing and adapter, to the needle airway of the blood bottle. The terminal segment

of a disposable fluid administration set is ideal because this plastic adapter fits snugly into the airway needle. If such is not available, the Luer-Lok tip of a 10-cc. syringe may be used as an adapter, and a piece of rubber tubing used to connect the adapter to a sphygmomanometer bulb. By this means, it is possible to deliver air under pressure over the blood without causing foaming. Additions to this basic equipment, including accessory bottles, filters, manometers, are frequently used in various institutions, but they are refinements, and are not absolutely essential. It is imperative, however, to have a hemostat at hand so that the pressure infusion can be arrested at any time. It is helpful to have pressure transfusion packs, consisting of the bulb, tubing, and adapter unit, and a hemostat available (fig. 4).

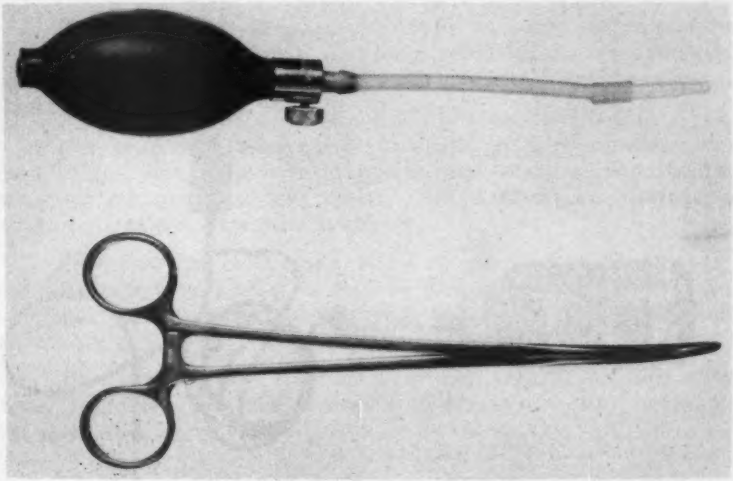

Figure 4. Contents of pressure transfusion pack.

An alternative method employs a syringe and a 3-way stopcock. The tubing from the bottle of blood is attached to the side arm; the tubing, plastic adapter, and 15-gage needle (the terminal segment of a disposable fluid administration set may again be used) are connected to the front; and a Luer-Lok syringe is connected to the rear aperture of the stopcock (fig. 5). This provides an effective means for rapidly infusing large quantities of blood. Occasionally, these stopcocks will admit air, in which case the syringe top should be tilted upward to allow air to rise.

It is important to realize that a pressure transfusion can be accomplished with apparatus which is readily available in any hospital.

THE RATE AND VOLUME OF BLOOD ADMINISTRATION

There is only one guidepost by which the speed of flow and the amount of blood given by artery may be determined. Blood is infused as rapidly as possible and in a quantity sufficient to bring about the desired blood pressure. There is no way to predict how much blood will be required. Once this result has been accomplished, the arterial transfusion should be terminated and maintenance blood therapy instituted by the conventional intravenous route. If inordinate amounts of blood are required and/or if a stable state is not reached, investigation should be directed toward the possibility of continuing hemorrhage or the presence of other etiologic factors potentiating the state of shock as discussed in the first section.

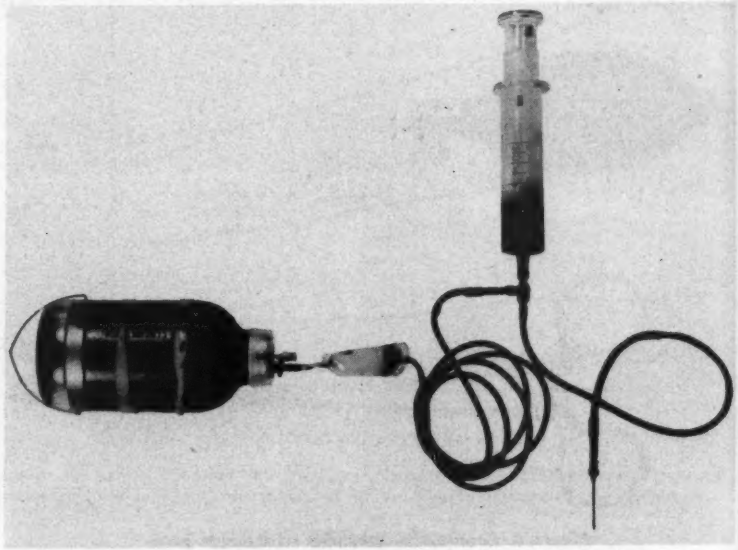

Figure 5. An alternative apparatus for pressure transfusion.

HAZARDS

The principal hazards are air embolism and permanent arterial damage with distal necrosis. Pressure infusion of air into the arteries can produce air embolism in the coronary arteries and death. Under no circumstances is it safe to give an arterial transfusion without constant surveillance by the physician in order to prevent this occurrence. If the physician must divert his attention, the pressure attachment should be disconnected. A hemostat must always be at hand so that the infusion may be immediately stopped. Arterial damage may result in gangrene of the extremity distal to the site of cannulation. The avoidance of

injury to the artery is the most important step in preventing thrombosis. By arresting bleeding by digital compression after withdrawal of the needle and by avoiding ligation of the artery, trauma to the vessel can be minimized.

SUMMARY

The basic technical considerations in arterial blood transfusion are:

1. *The condition of the patient.* Ascertain the primary and contributory causes of the shock and correct derangements of physiology.

2. *The artery.* Any artery is satisfactory. Cut down on the femoral or radial artery if necessary. Avoid ligation if possible. Compression hemostasis will usually suffice.

3. *The needle.* A 15-gage needle is preferred. It should be inserted so that the artery is cut longitudinally, not transversely, and fixed to the adjacent tissue for security, not by arterial ligature.

4. *The apparatus* need not be complex. A sphygmomanometer bulb, tubing, and adapter setup is attached to the airway needle. A 3-way stopcock and syringe may be used as an alternative method. A hemostat must be available.

5. *How fast and how much blood is given.* Rapidly enough, and in quantity sufficient to restore blood pressure, pulse rate, et cetera, to desired values. Switch to intravenous transfusion when stable.

6. *The hazards* are air embolism and arterial damage with distal necrosis. Both are avoidable if proper care and vigilance are exercised.

BOOK REVIEWS

Cardiac Therapy, by *Harold J. Stewart,* M. D., Associate Professor of Medicine, Cornell University Medical College, New York; Attending Physician, New York Hospital; Head of Division of Cardiology, Department of Medicine, New York Hospital-Cornell Medical Center. 622 pages; illustrated. Paul B. Hoeber, Inc., New York, N. Y., publishers, 1952. Price $10.

Cardiac therapy as prescribed and practiced by the author is completely outlined in this publication. Dr. Stewart has presented his personal experience in caring for the cardiac patient, but

unfortunately in doing so he has been repetitious in his description, so that at times it wearies the reader. This was probably done because the book is intended to be used as a reference for the therapy of a particular condition, and not for continuous reading.

The author's experience, both clinical and investigative, with the use of digitalis is presented in great detail. This portion of the book is highly recommended. Certain of his other ideas concerning therapy may not be accepted by most cardiologists. He still believes in the restriction of fluids with a low salt diet and is firmly convinced that strict bed rest is mandatory. He disregards the "armchair" treatment of congestive heart failure or the evil sequelas of bed rest.

The discussions of the treatment (including surgical methods) of rheumatic heart disease and congenital heart disease are sound and informative. Some cardiologists may not agree with the amount of penicillin recommended in the treatment of subacute bacterial endocarditis. Likewise, although the book is filled with the latest developments in the field, one finds that the use of benemid for the maintenance of higher penicillin levels is omitted, although carinamide is discussed.

The chapter on diseases which may have cardiac manifestations or simulate cardiac disease is magnificent for the orientation of the physician who is interested in cardiac diseases. The book will be most valuable to the general practitioner, for through its use he may gain from the experience of an outstanding authority, but it will be of only passing interest to the qualified cardiologist.—*Commander H. A. Lyons, MC, U. S. N.*

Electrotherapy and Actinotherapy, A Textbook For Student Physiotherapists, by *E. B. Clayton,* M. B., B. Ch. (Cantab.), Consulting Physician to the Physical Treatment Department, King's College Hospital, London. 2d edition. 452 pages; illustrated. Published in the United States by Williams & Wilkins Co., Baltimore, Md., 1952. Price $4.

The book is designed primarily for physiotherapy students and as such meets that need. It is divided into three main parts: Part I, which deals with electrotherapy; Part II, with actinotherapy; and Part III, treatments. Part I is subdivided into 17 chapters, the first being a clear and simple discussion of static electricity. The chapters on units and ionic theories and cells and cell batteries are well presented. The remainder of this part of the book is devoted to condensers, magnetism, meters, motors, currents, normal and denervated muscles, and diathermy. Part II is concerned with actinotherapy. Here the physics of infrared rays and ultraviolet light are explained along with their effects and technics for use. A classification of the methods of treatment and a discussion of some of the common conditions that respond to these methods of treatment are presented in Part III.

—*Lt. Col. R. C. Psaki, MC, U. S. A.*

STAB WOUND OF THE CEREBRUM

CHARLES R. REINERS, *Captain, MC, U. S. A.*

WILLIAM E. PHILIP, *First Lieutenant, MC, U. S. A.*

BECAUSE of the unusual circumstances surrounding the occurrence of a stab wound of the cerebrum, its manifestations, and the patient's dramatic recovery, the case is believed to be of sufficient general interest to justify preliminary reporting.

CASE REPORT

A 31-year-old sergeant presented himself on 2 January 1952 complaining of numbness of the right lower portion of his face and weakness and numbness of his right hand of 24 hours' duration. Questioning disclosed a head injury incurred at about 0500 hours on 1 January while at a New Year's celebration. He was aware of having been struck over the left side of his head, but had no knowledge of the instrument used. A friend who had been present volunteered that he thought the patient had been struck by a bottle. Although stunned for 5 or 10 minutes he had not lost consciousness or fallen to the floor, but had immediately lost the ability to move his right thumb and index finger. He remembered concentrating very hard immediately after the injury in an effort to make these movements. Following the episode he returned to the barracks and slept soundly until late in the afternoon. On awakening he noted persistence of the weakness of his right thumb and index finger as well as numbness of these fingers and the right side of his face. Late that day he was seen in his unit dispensary complaining of a headache and hang-over and he received symptomatic treatment. He returned once again to his barracks and slept until the morning of 2 January, when he noted extension of the numbness to the other fingers, palm, and dorsum of his right hand. Weakness was also present and his speech was now somewhat slurred. There was no weakness of his right leg.

Because of the progression of his symptoms he was referred to the 379th Evacuation Hospital where a careful history revealed that 13 years prior to admission he had incurred a skull fracture in an automobile accident for which he had been hospitalized for 1 month; and 9 years prior to admission he was hospitalized for 2 weeks with a possible skull fracture.

On admission he was alert and rational. A laceration 1.5 cm. long was observed over the left parietal region of his scalp.

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His pupils were regular and reacted well to light, and the optic disks appeared normal. A lower right facial palsy and weakness of his right hand were definite. The sense of touch was present over the right lower portion of his face and hand, although diminished, and pain was barely perceived. No weakness of his right arm or right leg was noted, nor were any pathologic reflexes elicited. Roentgenograms of his skull (figs. 1 and 2) disclosed a small pocketknife blade penetrating his left parietal bone and entering the cortex in a posterior and medial direction. The distal broken end of the knife blade appeared to be flush with the outer table.

Clinically his signs progressed with increased facial palsy, slurring of speech, and weakness and numbness of his right hand causing him to knock articles off the bedside table when he attempted to grasp them. Sixty hours after the stabbing, using an endotracheal tube and pentothal anesthesia, an incision was made in the line of the laceration with excision of the lacerated skin edges. An adequate purchase could not be obtained on the broken edge of the knife blade and a burr hole was placed immediately adjacent to it. Bone was removed with a ronguer until a dirty, blackened blade (fig. 3) could be freed and extracted. All damaged bone was then removed and the lacerated dura opened widely. Two small fragments of indriven bone and a small amount of necrotic cortical tissue were removed from the cortex. The wound was irrigated and closed.

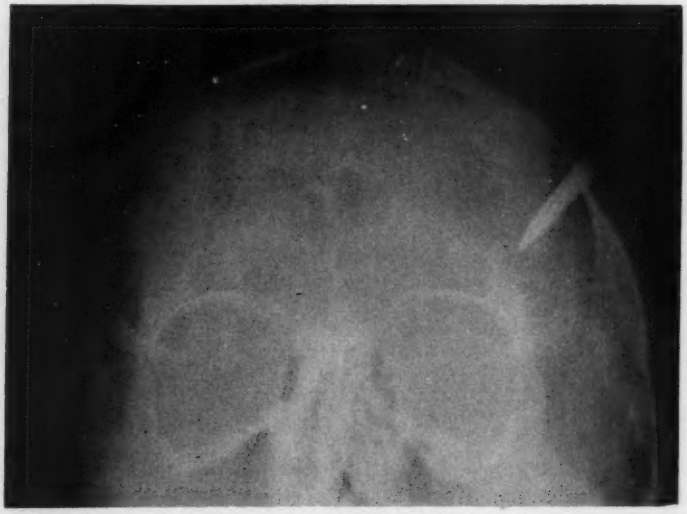

Figure 1. Anteroposterior view of skull.

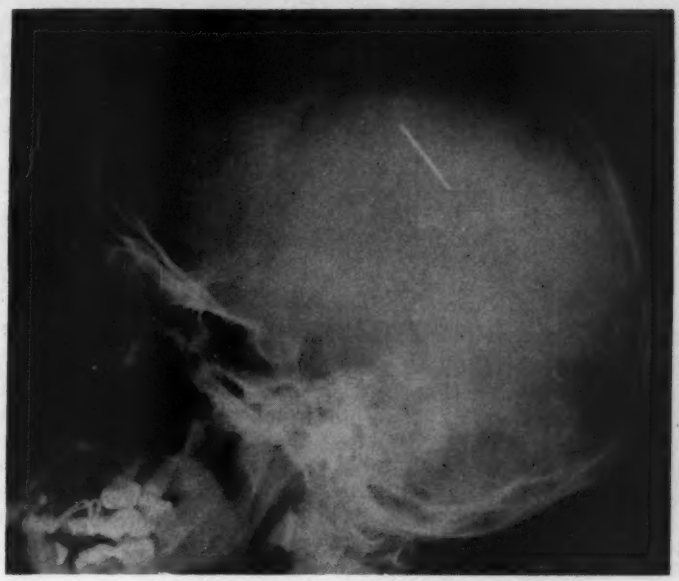

Figure 2. Laterial view of skull.

On the day of the operation he regained consciousness and voided, but catheterization was required on the first postoperative day and an indwelling catheter was required for 48 hours. Supplementary parenteral fluids were supplied only on the first postoperative day. Thereafter fluid intake was adequate. He was given 300,000 units of penicillin twice daily, 0.5 gram of streptomycin every 6 hours, and an injection of tetanus toxoid. From a high of 101° F. on the first postoperative day his temperature became normal on the fourth postoperative day and remained normal thereafter. Gradual improvement of his speech and right facial palsy occurred, becoming complete on the seventh postoperative day. The strength and sensation of his third, fourth, and fifth fingers quickly returned as did sensation of his hand. The power and co-ordination of his thumb and index finger returned slowly, despite his ability to perform all movements. He was unable to write or shave until 2 weeks after the operation. The acuity of pain and light touch sense remain diminished, but his proprioceptive sense was intact. When last heard from, 3 months after operation, he was asymptomatic, performing full duty, and had submitted an application for the grade of warrant officer.

DISCUSSION

Although no conclusions are as yet warranted, this patient demonstrated a gratifying defense against a potential infection

despite the time interval between the introduction and extraction of the foreign body. Although the value of antibiotics is not to be underestimated, we believe that emphasis should be placed on adequate débridement, particularly of indriven bone fragments because such fragments are found in a large percent of

Figure 3. Pocketknife blade, 1¼ inches long, removed at operation.

brain abscesses following penetrating wounds of the skull. The recovery of function that followed removal of the irritating stimulus, the absorption of blood clots, and the subsidence of edema were equally gratifying. Granted that sequelas may persist in this patient because of the severance of pathways in the posterior limb of the internal capsule, the fact that both motor function and sensation in the thumb and index fingers are grossly intact, though diminished, favors an optimistic prognosis.

BOOK REVIEW

Brain Mechanisms in Coronary Disease, Causation, Treatment, and Prevention, by *N. E. Ischlondsky,* M. D., Author of Brain and Behaviour; Protoformotherapy in Treatment and Prevention; Der Bedingte Reflex und Seine Bedeutung; Physiologische Grundlagen der Tiefenpsychologie, et cetera. With Two Appendices: 1. From the Conditioned Reflex to the Science of Brain Dynamics. Paper delivered at the XVIIIth International Congress of Physiology. 2. Reflexologic Bases of Personality. Paper delivered at the Ninety-Sixth Annual Meeting of the American Psychiatric Association. 171 pages; illustrated. Henry Kimpton, London, England, publisher, 1952.

Dr. Ischlondsky has written a most stimulating and informative monograph from a physiologic point of view. He stresses that at the basis of all behavioral reactions there is a mobile equilibrium between the two fundamental processes of the nervous system—excitation and inhibition. He clearly explains, documents, and clarifies the basic neurophysiologic concepts of the organism. The ideas introduced are refreshing, stimulating, and a definite contribution to the physician's understanding of his patient. I would recommend this book enthusiastically to all physicians, regardless of specialty.

—*Commander L. S. Madlem, MC, U. S. N.*

THE RELATION OF HYPERTENSION TO ARTERIAL ANEURYSMS OF THE BRAIN

BOYD K. BLACK, *Lieutenant Commander, MC, U. S. N.* (1)

SAMUEL P. HICKS, *M. D.* (2)

THE nature and origin of aneurysms of the arteries of the brain which are not obviously inflammatory have been the subject of many studies, but the cause of this not uncommon lesion has remained obscure. Although the concept that the principal lesion predisposing to aneurysm is a congenital defect in the muscular layer of a cerebral artery has been widely held, the trend has been to consider acquired lesions of the intimal elastic tissue as very important. The observations that medial defects are fairly frequent in normal cerebral arteries and that aneurysms are virtually absent in infants and children have been cited in support of this. The possible role of high blood pressure in producing these lesions has received some attention but the matter has remained controversial. It is useful, therefore, to note briefly some of the studies that bear on this problem of acquired disease, especially as they relate to hypertension and its associated cardiovascular lesions.

Fearnsides (3) considered the possible relation between "congenital" aneurysm and ventricular hypertrophy and concluded that although aneurysms were often found associated with cardiovascular disease, in many patients there was no evidence of hypertension clinically or at autopsy and that the degeneration of the cerebral arteries was no greater than would be expected in other subjects in the same age group in the general population. Abbott (4) found a high incidence of aneurysms in patients with coarctation of the aorta, but whether this is an association of 2 congenital vascular defects or the result of elevated blood pressure remains undetermined. Forbus (5), in a

(1) U. S. Naval Medical School, National Naval Medical Center, Bethesda, Md.
(2) The New England Deaconess Hospital and Harvard Medical School, Boston, Mass.

(3) Fearnsides, E. G.: Intracranial aneurysms. Brain 39: 224-296, Oct. 1916.

(4) Abbott, M. E.: Coarctation of aorta of adult type; statistical study and historical retrospect of 200 recorded cases with autopsy, of stenosis or obliteration of descending arch in subjects above age of 2 years. Am. Heart J. 3: 574-618, June 1928.

(5) Forbus, W. D.: On origin of miliary aneurysms of superficial cerebral arteries. Bull. Johns Hopkins Hosp. 47: 239-284, Nov. 1930.

study of the origin and mechanisms involved in the formation of "congenital" aneurysm, stated: "While the evidence at hand indicates that the muscularis defect constitutes the ground work for miliary aneurysm formation, there are convincing facts which cause us to regard the strength and persistency of the internal membrane as a factor of greatest significance in the development of anatomical aneurysms. While it is conceivable that small diverticula at the point of bifurcation may occur while the internal elastic membrane is still intact, it seems that no anatomical aneurysm can develop without disintegration of this membrane. In our study it has been possible to rule out all known causes of elastic tissue degeneration except the traumatic factor of overextension due to blood pressure. This we regard as the essential cause of the internal elastic membrane disintegration which we have found constantly in the miliary aneurysm."

Glynn *(6)* subjected a number of human circles of Willis, obtained at autopsy, to a pressure of 600 mm. of mercury. As long as the elastica remained intact, the vessels withstood the pressure and neither congenital nor artificially induced defects in the media caused the vessel to give way. Richardson and Hyland *(7)* observed that structural bifurcation defects of the media predisposed to aneurysms but that there was an additional acquired lesion which acted by weakening the elastica. They emphasized that if it were merely intravascular pressure one would expect aneurysms to be much more common in patients with arterial hypertension. Magee *(8)* concluded from a clinical study of 150 patients that neither physiologic nor pathologic high blood pressure caused aneurysmal rupture. Carmichael *(9)* considered the interrelation of congenital and acquired anatomic disease and believed that probably both lesions need to be present to cause an aneurysm. He did not implicate hypertension. Such diversified opinions by students of the disease suggest that a single causative factor in the genesis of aneurysms will not be found.

In a recent clinicopathologic study of the cause of cerebral hemorrhage *(10)*, the authors were impressed by an apparent frequent association of hypertension with cerebral arterial aneurysms in a small series of patients. As a result, we reviewed a series of

(6) Glynn, L. E.: Medial defects in circle of Willis and their relation to aneurysm formation. J. Path. & Bact. 51: 213-222, Sept. 1940.

(7) Richardson, J. C., and Hyland, H. H.: Intracranial aneurysms; clinical and pathological study of subarachnoid and intracerebral haemorrhage caused by berry aneurysms. Medicine 20: 1-83, Feb. 1941.

(8) Magee, C. G.: Spontaneous subarachnoid haemorrhage; review of 150 cases. Lancet 2: 497-500, Oct. 23, 1943.

(9) Carmichael, R.: Pathogenesis of non-inflammatory cerebral aneurysms. J. Path. & Bact. 62: 1-19, Jan. 1950.

(10) Hicks, S. P., and Black, B. K.: Relation of cardiovascular disease to apoplexy; review of 155 cases with autopsy. Am. Heart J. 38: 528-536, Oct. 1949.

100 cases of aneurysm studied at autopsy in relation to hypertension and cardiovascular disease. The principal aims of the study were to learn how frequently cardiac hypertrophy, cerebral arteriosclerosis, and especially clinically recognized hypertension are associated with aneurysm and to attempt to evaluate the importance of these factors. Some of the unsolved problems in the pathogenesis of aneurysms are indicated.

MATERIAL

The principal material used consisted of the autopsy reports of 100 patients who died with arterial aneuryrms of the brain. In most instances bleeding from the aneurysm or frank rupture was the primary cause of death. The autopsies were performed in the section on Pathologic Anatomy of the Mayo Clinic, Rochester, Minn.; the U. S. Naval Medical School, National Naval Medical Center, Bethesda, Md.; the New England Deaconess Hospital, Boston, Mass.; and Gallinger Municipal Hospital, Washington, D. C. The age, sex, heart weight, body weight, ratio of heart to body weight, history of hypertension, and the presence of sclerosis of the arteries of the brain were determined. A history of a consistent diastolic pressure in excess of 90 was considered to be hypertensive. A heart-to-body weight ratio greater than 0.5 percent was considered to represent hypertrophy.

RESULTS

The results are summarized in figure 1 and table 1. In figure 1 the percentage of patients who had cardiac hypertrophy, hypertension, and cerebral arteriosclerosis is compared with the incidence of hypertension in the general population *(11)*. Arteriosclerosis of the brain shows a steady increase with age as would be expected. Cardiac hypertrophy and hypertension run a close parallel to each other. The relatively greater percentage of cardiac hypertrophy than hypertension in each group is a result, for the most part, of failure to obtain a history of hypertension in that some of these patients were seen only in the terminal stage of their illness or after death had occurred. The fact that cardiac hypertrophy may rarely occur in the absence of high blood pressure or other demonstrable cardiovascular disease may account for an isolated example.

What is more notable is that there is both a positive and a negative correlation between hypertension and aneurysm. That is, there is definitely a high incidence of hypertension in patients with aneurysms, suggesting a cause and effect relation, yet in some patients with aneurysm there is no high blood pressure. On

(11) Robinson, S. C., and Brucer, M.: Range of normal blood pressure; statistical and clinical study of 11,383 persons. Arch. Int. Med. 64: 409-444, Sept. 1939.

the one hand, in the fifth and sixth decades (54 patients) the incidence of hypertension is 56 percent, significantly above the maximum incidence for the general population in that age group, which according to Robinson and Brucer *(11)* ranges to a maximum of 11.7 percent in men and 12.9 percent in women. In the third decade, 45 percent of 11 patients with aneurysm had a definite history of hypertension as contrasted with an incidence of about

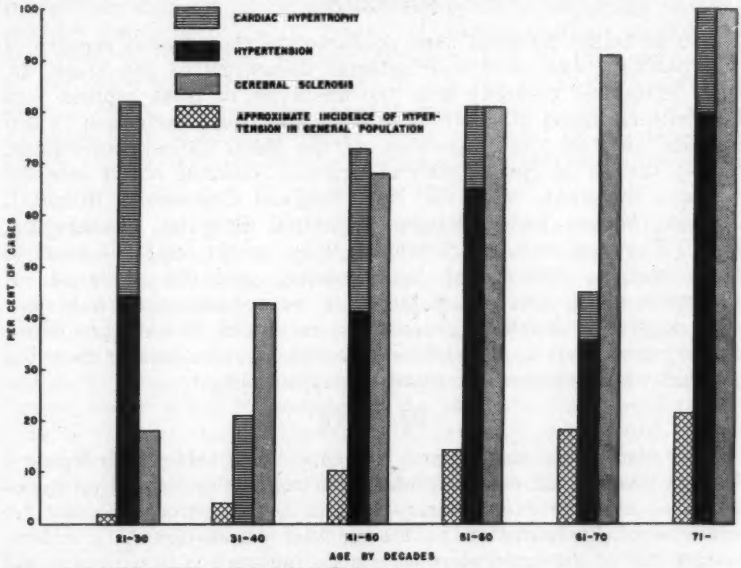

Figure 1. The incidence of hypertension, cardiac hypertrophy, and cerebral arteriosclerosis in a series of 100 cases of cerebral aneurysm compared with the incidence of hypertension in the general population.

2 percent in the general population for this group. On the other hand, a history of hypertension was absent in all of 14 patients in the fourth decade. Even assuming that the 3 patients who had cardiac hypertrophy had unrecognized high blood pressure, this incidence is far below that of other age groups in this series and shows that chronic hypertension cannot be an absolute requisite for the development of an aneurysm.

Because it is clear that hypertension is not a sine qua non of aneurysm formation, it can only be assumed that hypertension is frequently associated with aneurysm. There is, however, much evidence, as outlined above, that disease of the elastica is an essential factor in the genesis of aneurysms. It may be that the high blood pressure acting directly on the diseased vessels in some patients predisposes to aneurysm formation, especially if

TABLE 1. *Summary of cardiovascular findings in 100 cases of aneurysm*

Age (years)	Number	Cardiac hypertrophy	Hypertension	Cerebral arterio-sclerosis	Rupture of aneurysm	Ratio of women to men
21-30	11	9	5	2	10	2:9
31-40	14	3	0	6	12	10:4
41-50	22	16	9	15	17	9:13
51-60	32	26	21	26	26	15:17
61-70	11	5	4	10	9	5:6
Over 70	10	10	8	10	3	8:2
Total	100	69	47	69	77	49:51

a defect of the muscularis is present. On the other hand, it is possible that the hypertensive state and the development of aneurysms are independent expressions of an underlying vascular disease process and have no cause-and-effect relationship with each other.

The incidences of cerebral arteriosclerosis and rupture of the aneurysm do not parallel each other or the incidence of hypertension. The significance of rupture in relation to high blood pressure and the fundamental pathology of aneurysm formation is unsolved.

SUMMARY

In 100 cases of intracranial arterial aneurysm, a history of hypertension was found in 47 percent of the total and 69 percent had cardiac hypertrophy. Of 54 patients in the fifth and sixth decades, 56 percent had hypertension. These incidences of high blood pressure are significantly higher than that reported in corresponding groups of the general population.

Notwithstanding, there was a substantial number of patients with aneurysms who had no evidence of hypertension or heart disease. It was concluded that the incidence of hypertension is significantly increased in persons with aneurysm but that hypertension is not an essential factor in the genesis of aneurysms. Hypertension and aneurysms may have a common underlying vascular disease process or processes, yet may not always be related as to cause and effect.

HYPERPLASIA AND HYPOPLASIA OF THE DENTITION

PETER F. LABAND, *D. D. S.* (1)

DEVELOPMENTAL anomalies of the dentition, especially hyperplastic conditions, are frequently found and may be classified as (1) predeciduous dentition; (2) supernumerary deciduous or permanent teeth if they have the form and size of a normal tooth or group of teeth; and (3) accessory teeth if they are of abnormal form and size (2, 3).

Considering that we have to deal with 52 individual organs of complicated structure which develop in a time span between intrauterine gestation to about 25 years of age, it is astonishing that we do not find more aberrations in the complex process. There is another group of anomalies which has given rise to some controversy: postpermanent teeth or third dentition. The reason for the difference of opinion seems to be that these teeth are often discovered only after the permanent teeth have been removed. It is then hardly possible to decide whether they are supernumerary teeth of the permanent dentition, caused by hyperactivity of the dental lamina at the time of the formation of the permanent teeth, or a hyperactivity of the lamina over a longer period after the formation of the permanent teeth has been accomplished. This time factor is the only guide to show us whether we are allowed the use of the term third dentition at all. Reports in the literature describe supernumerary teeth which, judging from the roentgenograms, were actually buds of postpermanent teeth situated between the roots of fully developed permanent teeth (4).

The following two cases of partial third dentition have been observed during routine roentgenographic examinations. In both patients we found a well-formed complement of permanent teeth with fully developed roots. The postpermanent group of teeth seen in the roentgenograms was still in the formative state and beginning to cause deviation of the roots of the permanent teeth by their growth. In these cases we think we are right in using the

(1) Dental Clinic, Camp Stoneman, Calif.

(2) Thoma, K. H.: Oral Pathology. 2d edition. C. V. Mosby Co., St. Louis, Mo., 1944.

(3) Adelstein, C. S.: Supernumerary teeth. Am. J. Orthodontics 29: 654-657, Nov. 1943.

(4) Brodie, A. G.: Two unusual cases of supernumerary teeth. Angle Orthodontist 6: 209-211, July 1936.

term postpermanent or third dentition because the group of teeth observed developed after the permanent teeth had completed their growth.

CASE REPORTS

Case 1. A 20-year-old man came to this clinic for fillings. As far as he remembered, he had a full complement of deciduous teeth and the eruption of his first and second dentition was normal. In the buccal fold on the alveolar process in the region of teeth 28 and 29, a cherry-sized bony protrusion which was indolent and showed no signs of inflammation, was seen. Dental roentgenograms showed teeth 3, 14, and 30 missing (extracted) and 4 tooth buds with fully developed crowns and beginning development of roots below teeth 20 and 21. Tooth 21 was tilted distally in its axis. Under teeth 28 and 29 there were 2 more tooth buds with no roots and their crowns not fully developed. Tooth 28 was tilted distally. Because the roentgenograms (fig. 1) showed that these growing tooth buds had already started to crowd and displace the adjacent permanent teeth, it was considered advisable to remove them.

On 29 November 1951 a semilunar incision was made at the gingival margin of the buccal aspect of the right mandible from teeth 27 to 30. A mucoperiosteal flap was elevated, revealing a bony protrusion. The outer cortex was chiseled away, disclosing the fully developed crown of a small cuspid. This was removed. Behind it, in a second bone crypt, there was a crown of the same type enveloped in a follicular sac. This also was removed. The 2 crypts were inspected and found free of bone and soft tissue debris.

On 30 November a semilunar incision was made on the lingual aspect of the alveolar process reaching from teeth 19 to 23, with the base toward the floor of the mouth. A mucoperiosteal flap was elevated. The freed bone showed no protrusion. The cortical plate was removed revealing the crowns of 2 tooth buds. These were removed. Anterior and slightly superior to the 2 crypts, a third bud was found and removed. The fourth bud which, according to the occlusal film, was located more buccally, could be seen wedged between the diverging roots of teeth 20 and 21, too deep to be reached from the lingual aspect. For this reason a semilunar incision was made at the buccal aspect from teeth 19 to 22. A mucoperiosteal flap was elevated and a small window cut into the cortex between teeth 21 and 22. The tooth bud was found, pushed into the lingual cavity, and removed. Bone and soft tissue debris was removed from the crypts. Teeth 20, 21, 28, and 29 were tested 2 weeks later and found to be vital.

Figure 1. Intra-oral roentgenograms showing tooth buds of third dentition in mandible.

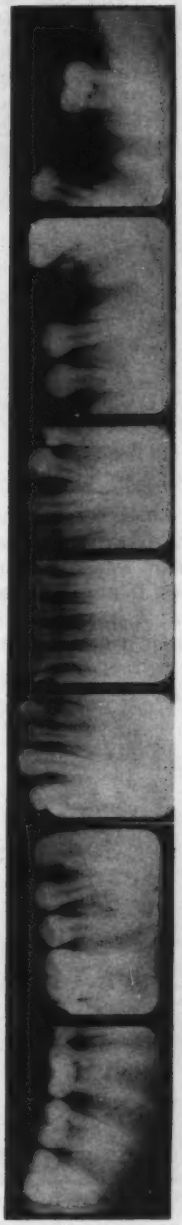

Figure 2. Intra-oral roentgenograms showing tooth buds of third dentition in mandible.

Case 2. A 19-year-old man came to this clinic on 21 February 1952 for treatment of a toothache in tooth 30, which had a periapical abscess. In routine examination, including roentgenograms, findings similar to those in case 1 were observed. There was deep caries in tooth 30. The roentgenograms showed: a tooth bud which developed only the crown in the region of teeth 20 and 21 between the lower third of the roots; tooth 21 was tipped distally, the apex pointing mesially; and at the apex of tooth 28, a corresponding tooth bud which had pushed the root of tooth 28 mesially (fig. 2).

On 27 February a semilunar incision was made in the lingual aspect of the right mandible reaching from teeth 26 to 30, with the base of the flap toward the floor of the mouth. A mucoperiosteal flap was elevated and the cortical plate was removed with a chisel and bur. The tooth bud was located between the root of teeth 28 and 29 and was removed with the follicular sac and pulp tissue which separated from the crown when removed. A similar operation was performed on the left side. Teeth 20, 21, 29, and 30 were tested after 10 days and found to be vital.

Comment. In both patients symmetrical supernumerary teeth below the same group of teeth which had developed after the second dentition was completed were found. Only the crowns were developed, but it could be expected that in due time the growth would have been completed followed by displacement and lesion of the permanent bicuspids.

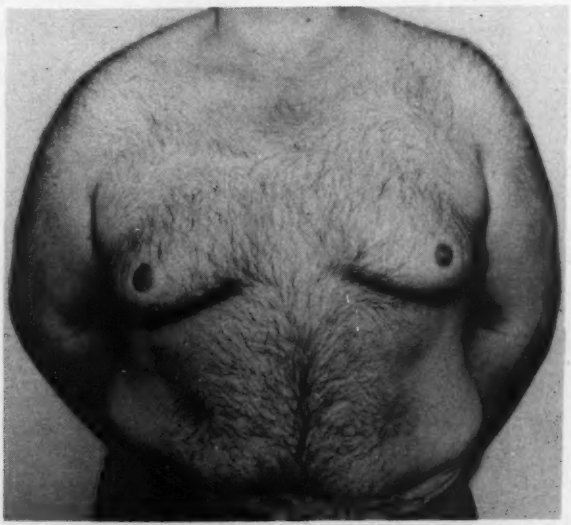

Figure 3. Patient with constitutional obesity and gynecomastia.

Figure 4. Teeth of patient 3.

The patient described below had a condition which was the opposite of that of patients 1 and 2, a dental underdevelopment, or ectodermal dysplasia. Many variations of this condition from the absence of a single tooth or group of teeth to anodontia and finally to anhidrotic ectodermal dysplasia with anodontia have been observed. All these aberrations appear to be inherited and can therefore show many variations according to the combination of genes contributed by each parent *(5)*. Endocrine disturbances may also be causative factors. Downs *(6)* found in a survey of 647 patients that the percent of dental anomalies in patients with endocrine disturbance is higher than normal but not specifically related to any type or group of them.

In my patient both factors had to be considered but the preponderance of the evidence pointed to a hereditary cause.

Case 3. A 22-year-old man came to this clinic to obtain a prosthesis. He had been obese since childhood. He developed normal deciduous teeth, but shed only teeth 8, 9, 20, 27, and 29. One sister who was also obese had "exactly the same teeth as he had." His father and one other of his 6 sisters were obese. The patient who was 70 inches tall weighed 240 pounds (fig. 3). The hair of his scalp was fine and sparse as were his eyebrows but he had a rather heavy growth of soft hair on his chest and back for a fair-haired man. His teeth 3, 8, 9, 14, 19, 27, and 30 were

(5) Everett, F. G.; Jump, E. B.; Sutherland, W. F.; Savara, B. S.; and Suher, T.: Anhidrotic ectodermal dysplasia with anodontia: study of two families. J. Am. Dent. A. 44: 173-186, Feb. 1952.

(6) Downs, W. G., Jr.: Studies in causes of dental anomalies. J. Dent. Research 8: 367-379, June 1928.

permanent but 4 to 6, 11 to 13, 20 to 27, and 29 were deciduous (fig. 4). Dental roentgenograms (fig. 5) confirmed the clinical findings and showed permanent tooth 27 to be impacted.

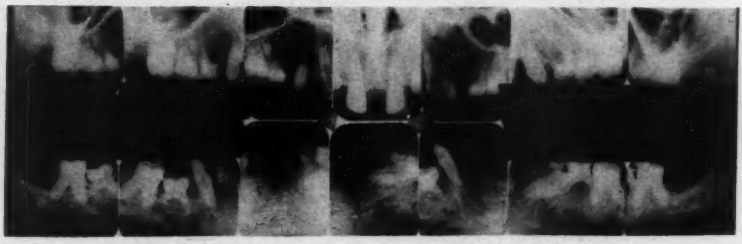

Figure 5. Intra-oral roentgenograms of patient 3.

The family history suggested a hereditary factor, and the patient's physique suggested an endocrine disturbance. For this reason the patient was examined in the endocrine clinic but nothing of importance was discovered. The laboratory examinations included a hemogram, serologic test for syphilis, urinalysis, blood sugar, glucose tolerance test, serum cholesterol, serum phosphorus, serum calcium, total serum protein, 17-ketosteroids, a BMR, and an examination of the sperms. All tests were within normal limits. Roentgenograms of the chest, skull, and wrist for bone age were normal. The obesity was classified as exogenous.

All of the patient's teeth were removed. Prostheses were constructed. The esthetic and functional effect was very satisfying to the patient.

SUICIDAL GESTURES IN OCCUPATION PERSONNEL ON OKINAWA

LUDWIG G. LAUFER, *Captain, MC, U. S. A.* (1)
DANIEL H. CASRIEL, *Captain, MC, U. S. A.* (1)

FEW physicians have adequate specialized psychiatric training, but all medical officers are called on to manage psychiatric patients from time to time. The purpose of this article is to help the medical officer render the correct evaluation and make the proper disposition of those patients in whom the problem of suicide arises. The physician should consider himself personally and solely responsible for the patient's total welfare only if the patient (1) is psychotic, (2) is overwhelmed by an obsessive-compulsive neurosis, (3) is neurotically depressed, or (4) has a severe mental deficiency. Patients who do not fit into one of these categories must assume responsibility for their own behavior, and it is the responsibility of the medical officer to return such patients to duty with the necessary explanation to the soldier's administrative superiors and to make recommendations regarding the amount of responsibility the patient must assume for himself. The commanding officer is thus aided in making the final disposition.

On Okinawa there is the unusual opportunity for observing patients over a longer time than might be possible in other theaters because most of the occupation personnel remain on the island for from 18 to 36 months. We are the only psychiatrists working in the only hospital here for the occupation personnel. Over a period of 21 months several patients who have made suicidal attempts or gestures were treated. From this experience a method of evaluating and disposing of such patients has been developed and has been amply tested. To date no patient has actually committed suicide, although some have made a second suicidal gesture. Because there is always the element of calculated risk, it is possible that some day, some ex-patient may go on to suicide, but the assumption of this risk cannot be shirked.

According to our definition, a person has made a suicidal attempt if he has a real desire to die at the time he commits the

(1) Ryukyus Army Hospital, Okinawa.

act, and a suicidal gesture is made by a person who does not really wish to die at the time he performs the act. Suicidal gestures may be a way of releasing tension but they are better understood as histrionic, attention-getting devices, or a passive-aggressive use of a self-inflicted wound to control the environment and to prevail on others to do what the patient desires. Disposition of the patient depends on the psychiatric state and not on the severity of the injury or whether the act was a suicidal attempt or gesture.

Several key questions should be asked routinely when talking to the witnesses who accompany the patient. It is important to determine what happened, when it happened, where it happened, and, if possible, why it happened. Was the patient under the influence of alcohol or drugs when found? If there is any suspicion of alcoholism, a blood alcohol test should be made. The names, addresses, and telephone numbers of the persons interviewed should always be obtained in case further investigation is necessary. It is especially important to get this information if the patient is unconscious, too depressed or withdrawn to communicate, claims amnesia for the event, or when he is so unco-operative that he displays an attitude of "figure it out for yourself." Even more so than in general medicine, the taking of a careful history will give the diagnosis. The patient's reason—the stresses which influenced him at the moment of his act—must be evaluated by the physician. He must be alert for evidences of psychosis, obsessive-compulsive ideation or behavior, neurotic depression, or obvious mental deficiency.

Before a final diagnosis is made, it is frequently necessary to contact the patient's superior officer for verification of alleged facts or evaluation of the patient's personality as observed in his organization. Some of the questions that should be answered are: How long has the soldier been in the organization? What is his disciplinary and medical record? How often and how much does he drink? What is his general behavior—does he get into frequent fights; does he "blow his top" easily; does he mix with the other men or is he seclusive; what does he do in his spare time; and what are his habits? How is his mood—has it recently changed; has he appeared to be sad or depressed; does he do a lot of daydreaming or anything peculiar; has he received bad news lately; and has he appeared frustrated or asked for a transfer or shown any resentment about his duties? What is his intelligence level? What is his character and efficiency rating? Is he regarded as a good soldier? This information will often make an evaluation of a case much easier and will, in addition, indicate leads to follow if re-examination of the patient is necessary. Sometimes this information can be obtained from those who bring in the patient. If the above information is not obtained, the clue

to diagnosis may be missed. In certain instances it may be wise to hospitalize the patient for further observation or until the essential data can be collected.

DIAGNOSIS AND DISPOSITION

Psychotic patients. The types of psychotic patients most frequently seen in military service as the result of self-harm are the schizophrenics and the alcoholics. Patients with psychotic depression are seen less frequently because this condition does not usually occur in patients under 40 years of age. Psychotic behavior and a history of alcoholism over a period of months or years with or without previous episodes of delirium tremens, delusions, or hallucinations will suggest a diagnosis of psychosis due to chronic alcoholism. The psychosis of the chronic alcoholic may persist for days, weeks, or months. Such a patient may have amnesia for some or all of his psychotic activities when he is finally free from the effects of alcohol. Even if such a patient makes a serious suicidal attempt, he should be returned to duty when he is no longer suffering from the effects of his alcoholism with a recommendation for an administrative discharge for his undesirable character. His commanding officer should be apprised of the possibility of another suicidal attempt if the man continues to drink. Psychotic behavior is seen in persons suffering from acute alcoholism but who are not chronic alcoholics. This condition will clear up in 24 hours. The patient who behaves in a disturbed manner resembling psychosis because of acute alcoholism should be returned to duty when sober. It is usually recommended to his commanding officer that he not be punished for his first suicidal gesture, although there are no medical contraindications for any disciplinary action as a result of his drunkenness.

Obsessive-compulsive patients. Only one of our patients had an irresistible compulsion to attempt suicide. Several patients were seen who had obsessive preoccupation with suicidal thoughts but who had not made a suicidal gesture or attempt. When it was believed that their obsession would not lead to compulsive suicidal attempts they were returned to duty. They were made to realize that they were mentally responsible and any foolish action on their part might result in severe disciplinary action. They were encouraged to return to the clinic for psychotherapy. Those who were emotionally disturbed by their thoughts returned and were helped by the therapy given. Those who used their verbalizations (of an obsession to die) for secondary gains either did not return or soon lost interest in therapy.

Patients with neurotic depression. Moderate and severe neurotic depressive reactions resulting in a suicidal attempt or gesture were caused by severe external stress, marked predisposi-

tion, or a combination of both. A neurotic depression may occur at any age. An acute severe stress such as a "Dear John" letter (2) or the loss of loved ones may lead to a suicidal attempt or gesture in a predisposed person. This type of response is called a "reactive depression" or an "acute situational maladjustment." A general dissatisfaction with his position in life, unresolved frustrations, and various chronic emotional stresses are usually evident in the history of the chronically depressed patient. Although depressive features are present in most patients examined after a suicidal attempt or gesture, most patients exhibiting depressive features are not truly neurotically depressed. Perhaps only 1 or 2 out of 10 "suicidal" patients are properly so diagnosed. The others are primarily immature and inadequate persons. If a working diagnosis of neurotic depression is made, the examiner should not hesitate to hospitalize the patient, if necessary, for further treatment or observation. Usually a neurotically depressed person will show signs of psychomotor retardation and a depressed affect. He will not push for secondary gains with such threatening statements to the doctor as, "If you don't get me out of the service, I'll kill myself." Rather he is worried over a particular problem which he usually has held for many weeks or months and which he has been unable to resolve. In addition, there are marked feelings of guilt, inadequacy, and unworthiness.

Mental deficiency. Because they are usually screened out before entering the Army and especially before they go overseas, we did not see any severe mental defectives. Mental deficiency per se is not a predisposing cause for suicide, but a mental defective under stress is likely to develop psychotic behavior and/or make a suicidal gesture or attempt. The mental defective who manages to remain in the Army has usually attained a high enough mental level to be responsible for his actions. In most cases, however, mental deficiency is a mitigating factor in the disposition of the patient. These soldiers are recommended for administrative separation from the service and are not recommended for disciplinary punishment.

Most of the patients seen had made their suicidal attempts as a result of immaturity reactions. All these patients were returned to duty. Glass (3) briefly but adequately defined an immature soldier as "one characterized by a retarded emotional development which is not commensurate with chronological age and physical growth. The result is an individual who exhibits ineffectual, childish, or adolescent behavior when exposed to slight or moderate stress." Immaturity reactions include emotional in-

(2) Letter telling him his fiancée was married or in love with another man.

(3) Glass, A. J.: Medical evaluation of the noneffective soldier. Surgeon's Circular Letter, FECOM 6: 74-79, Apr. 1951.

stability, passive-aggressive, passive-dependency, and aggressive reactions and alcoholism. Simple drunkenness is also included in this group.

A few of our patients were classified as having a pathologic (or psychopathic) personality. The inadequate, the schizoid, the antisocial, the asocial, and the sexually deviated personality are included. This type of patient not infrequently resorted to a suicidal gesture because of an acutely difficult situation following his own misconduct. Patients with either an immature or a pathologic personality are mentally responsible for their actions and therefore subject to administrative action—either punishment or discharge. All these men should be returned to duty. The recommendation sent with each man depends on the individual circumstances. The most important factor to be considered is the severity of the immature or pathologic personality.

When a "suicidal" patient is discharged to duty, a letter should be sent to his commanding officer. If the soldier is likely to reach his commanding officer before the letter, the commanding officer is notified by telephone or messenger of the soldier's mental status (responsibility) and the recommended disposition. This is followed by a letter written in nontechnical language. Such action makes it easy for the commanding officer to decide on the proper disposition.

TYPICAL LETTERS

Example 1. This soldier has no mental illness. He was drunk at the time of his suicidal act and is very chagrined over his actions. He could give no reason for his act nor could any be found. It is unlikely that this act will recur. He was warned that this type of behavior may result in severe disciplinary action. It is suggested that no additional (or minimal) disciplinary action be taken because of the suicidal gesture.

Example 2. This soldier has an immature personality. Like a child who pounds his head against a wall when he does not get his way, this soldier harms himself. He does not really wish to commit suicide and his act is only an immature response to his frustration, resembling a temper tantrum. Because his basic personality has not changed, it is possible that he may again resort to acts of self-harm. Such acts could accidentally become serious and even lead to his death, but such a result is unlikely. He is mentally responsible for his actions. He is cleared by this section for any disciplinary action that is deemed advisable by his commanding officer.

Example 3. This soldier is an immature person who reacts to minimal stress in an emotionally unstable manner. He is responsi-

ble for his actions, but his personality is so immature that further gestures of a suicidal nature can be anticipated. It is doubtful whether these will be successful. Disciplinary action will not reform him. Psychiatric treatment is not indicated. His value to the service is nil. It is therefore recommended that he be discharged from the service as unadaptable (or unsuitable or unfit).

Example 4. This soldier is responsible for his actions and is therefore subject to disciplinary action. He is not mentally ill, but his personality is so defective (inadequate, alcoholic, or immature) that his further retention in the service is not advised. Although he has the capability of adjusting to military life, he will probably not adjust. It is therefore recommended that he be discharged from the service as undesirable.

Example 5. This soldier had an illness which led to his suicidal attempt. This condition has improved with treatment. He is being returned to duty. He will continue to receive treatment in the neuropsychiatric outpatient clinic at regular intervals. It is recommended that no disciplinary action be taken for his suicidal act.

SUMMARY

Before an evaluation and disposition of a suicidal patient is made, the witnesses, associates of the patient, and his superiors should be questioned. The medical officer should not be emotionally influenced in his evaluation. The disposition of the patient is determined by the underlying mental or personality condition rather than by the severity or nature of the suicidal attempt or gesture. The physician is primarily responsible for the future of the patient only when the patient is psychotic, has an obsessive-compulsive neurosis, a neurotic depression, or is mentally defective. It is the medical officer's responsibility to return all other patients, without exception, to their organization with recommendations for administrative or disciplinary action.

THE TECHNIC OF DISCOGRAPHY

PETER G. GLEASON, *Lieutenant, junior grade, MC, U. S. N. R. (1)*

DISCOGRAPHY is the examination of intervertebral disks by injecting contrast media directly into the disk spaces. The procedure was first reported by Lindblom *(2, 3)*. Wise et al. *(4)* and Gardner et al. *(5)* have used a modified Lindblom technic. Without the use of fluoroscopic control and with the patient in a modified knee-chest position achieved by having the patient bend over a table equipped with a knee rest, a 2½-inch, 21-gage spinal needle is inserted between the spines of the vertebras adjacent to the suspected disk. Then, a 3½-inch, 26-gage spinal needle is passed through the 21-gage needle into the disk space. The position of the needles is checked by anteroposterior and lateral radiographs, and from 0.5 to 2 cc. of 35 percent iodopyracet compound solution is injected directly into the nucleus pulposus or into the posterior portion of the annulus fibrosus. The presence or absence of reproduction of the patient's symptoms is noted, and anteroposterior and lateral radiographs are immediately made. For very large patients a 4½-inch, 26-gage needle is used.

Smith et al. *(6)* have been using a posterolateral approach with a single, long, 20-gage spinal needle. The insertion of the needle is accomplished with the patient lying on his side, and without the aid of fluoroscopy. The entrance of the needle into the posterior longitudinal ligament is felt by the operator. Following this sensation, the needle is inserted an additional 1.5 cm. so that its point will lie near the center of the disk. The position of the needle is checked by anteroposterior and lateral radiographs. A water soluble contrast medium is then injected, and anteroposterior and lateral radiographs are made.

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(1) U. S. S. *Consolation.*

(2) Lindblom, K.: Diagnostic puncture of intervertebral disks in sciatica. Acta orthop. Scandinav. 17: 231-239, Aug. 1948; Nord med 38: 1256, 1948.

(3) Lindblom, K.: Technique and results in myelography and disc puncture. Acta radiol. 34: 321-330, Oct.-Nov. 1950.

(4) Wise, R. E.; Gardner, W. J.; Hughes, C. R.; and Weiford, E. C.: X-ray visualization of intervertebral disk. Modern Medicine 20: 104-112, June 1952.

(5) Gardner, W. J.; Wise, R. E.; Hughes, C. R.; O'Connell, F. B., Jr.; and Weiford, E. C.: X-ray visualization of intervertebral disk; with consideration of morbidity of disk puncture. Arch. Surg. 64: 355-364, Mar. 1952.

(6) Smith, G., et al.: Discography in diagnosis of herniated intervertebral disc. A. M. A. Annual Convention, Space 378, June 1952.

Erlacher (7) has used the posterolateral approach. He described the use of a double cannula needle 12 cm. long. Without the aid of fluoroscopy and with the patient either on his side or in a sitting position, the skin and adjacent deeper structures are infiltrated with procaine solution. Anteroposterior and lateral radiographs may be made with the needle in place for orientation purposes. The 12-cm. needle is inserted "½-inch lateral to the spinous process and immediately above the upper margin of the spinous process of the adjacent lower vertebrae and directed upward and medially." For the lumbosacral disk the needle is inserted a little more cephalad and directed downward and medially in order to allow for the angulation of the lumbosacral disk. The position of the needles is checked by anteroposterior and lateral radiographs; 1 or 2 cc. of a water soluble contrast medium is injected; and anteroposterior, lateral, and oblique radiographs are made.

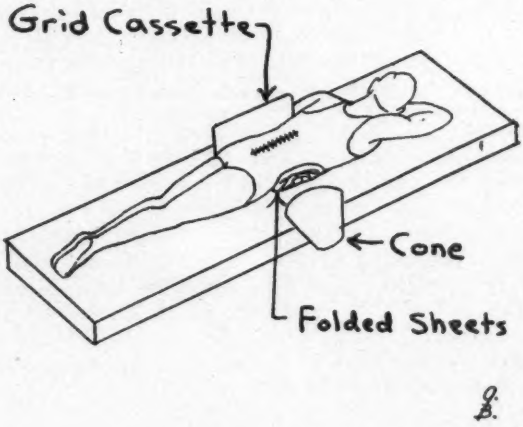

Figure 1. Position of patient.

The technic used at this institution is similar to that described by Lindblom and uses an almost directly posterior approach. A long 26-gage needle, which is passed through a short 21-gage needle, is used. A roentgenologist and a technician are the only personnel required, although the assistance of another physician is helpful. A preliminary lateral radiograph of the lower lumbar spine is made with the patient lying on 3 folded single sheets which serve to straighten out the lordotic curvature (fig. 1).

(7) Erlacher, P.: Nucleography. J. Bone & Joint Surg. 34: 204-210, May 1952.

The centimeter measuring rod (fig. 2) is previously placed over the patient's lower lumbar spinous processes, adjacent to the skin. The centimeter markings on the rod will be distorted on the film to about the same extent as the distances between the skin and the centers of the disks. When completely inserted, the

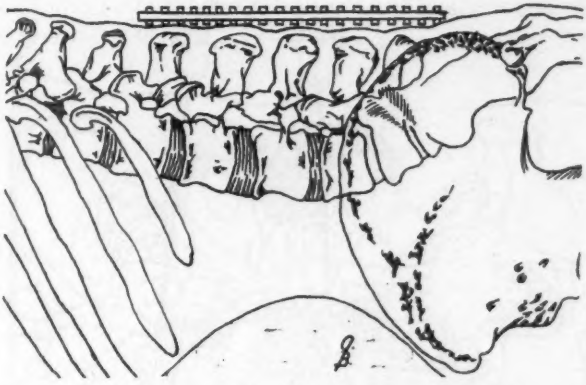

Figure 2. Measuring rod, lumbar spine, and abdominal compression with folded sheets.

26-gage needle should lie in the center of the disk. The length of the 26-gage needle required can be determined, by adding ½ inch to the corrected distance between the skin and each disk center. The ½-inch addition is to allow for that portion of the needle which lies above the skin inside the butt of the 21-gage needle. In adults, a needle 3½- or 4-inches long is usually required. If desired, the lengths required for the 21-gage needles, to barely reach the meninges, can also be determined on the preliminary film, the distance varying from 1¾ to 2½ inches. Because the measuring rod lies on the skin, the inferior border of the rod represents the surface of the skin on the radiograph. If it is difficult to see the rod on the film, one side of the film emulsion in the region of the rod can be scraped off while the film is wet. The preliminary film can be made 1 or 2 days in advance if desired.

An hour before the discograph is to be made, the patient is given 100 mg. of secobarbital sodium and 30 min. before the examination he is given from 10 to 15 mg. of morphine sulfate. When he arrives for the discography, he is given an intradermal

sensitivity test for the water soluble contrast medium to be used (35 percent iodopyracet compound has been found satisfactory). He then assumes the prone position again with the sheets folded under his lower abdomen, and is prepared and draped with sterile towels by the technician while the physician arranges his needles, syringes, solutions, et cetera. With the left hand operating the fluoroscope and the right hand sterile, the physician places the point of a needle attached to a sterile syringe full of 1-percent procaine on the patient's skin over the site where the 21-gage needle is to be inserted. This is done under fluoroscopic visualiza-

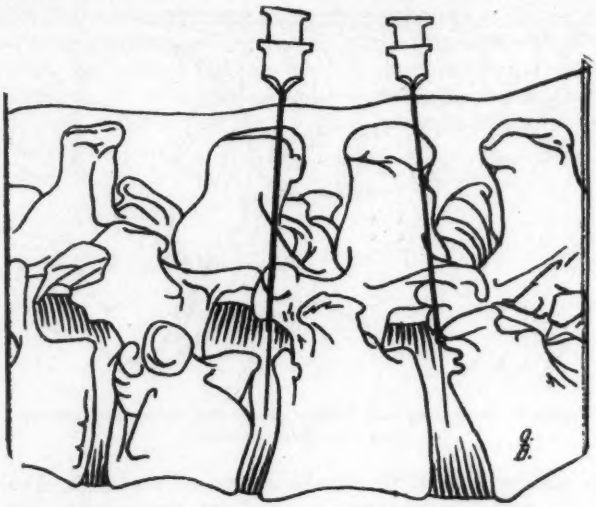

Figure 3. Lateral view showing needles in place.

tion (figs. 3 and 4). Usually the best place for the insertion is just lateral to the spinous process of the vertebra above the disk and just caudal to its lamina, as visualized in the anteroposterior view. It is helpful if the operator first studies the patient's routine lumbosacral radiographs, in order to familiarize himself with the shape and position of the patient's lamina, spinous processes, and disk spaces. A study of the lateral view taken for measurement will enable him to estimate the degree of cephalic or caudal angulation which may be required in inserting the needles (fig. 4).

Then with the aid of a red flashlight held by the technician, a wheal is made in the skin where the needle point lies, and the adjacent deeper structures are infiltrated. If desired, the sites of insertion for all 3 disks can be prepared at one time. Following a negative skin test, the 21-gage needle is inserted with slight

medial angulation through the wheal for a distance of from ½ to 1 inch. The position and direction of the needle is checked fluoroscopically. If the point of the needle is obscured by its butt, traction on the adjacent skin (outside of the fluoroscopic field) will change the angulation of the needle so that the location of its point can be visualized. If it is found to be pointing in the wrong direction, redirection should not be attempted when the needle is completely inserted because of the possibility of tearing the meninges. A slight cephalic direction is required for the disk below the third lumbar vertebra, a directly anterior direction for the disk below the fourth, and a slightly caudal direction for the lumbosacral disk.

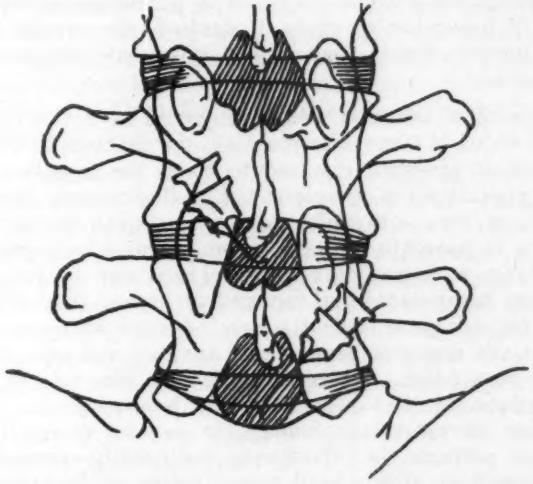

Figure 4. Anteroposterior view showing needles in place.

After the 21-gage needle is inserted completely, the 26-gage needle is passed through it into the disk. Passage through the meninges is felt by the operator as a slight pop. The passage through the posterior longitudinal ligament is felt as a stronger pop. Passage through the annulus fibrosus gives a rather firm rubbery fibrous sensation. The position of the needles is checked fluoroscopically in the anteroposterior projection. In spite of the small size of the 26-gage needle, its distal end can be seen fluoroscopically because it is viewed almost on end.

A 2-cc. syringe containing the contrast medium is attached to the 26-gage needle. Although this can be done with one hand, it is recommended that the operator put a sterile glove on his left hand, previously used to operate the fluoroscope, in order to

be able to use both hands for the attachment of the syringe and for the injection. If an assistant is present, much time can be saved by having him attach the syringe to the needle, and perform the injection because he can keep both hands sterile throughout the entire procedure. From 1 to 2 cc. of the contrast medium is injected through the 26-gage needle and the patient is asked to describe any sensations he may have. Leading questions are avoided. The contrast medium is observed fluoroscopically and if found to be adequately visualized, a lateral radiograph is made using a stationary grid. This may be done either before or after the withdrawal of the needles. Then with the needles withdrawn, anteroposterior and oblique radiographs are made with a spot film device. The prone position facilitates the turning of the patient for oblique views. The procedure is repeated for the other 2 lower lumbar disks if desired. We usually inject all 3 lower lumbar disks, even if the symptoms suggest a lumbosacral lesion.

The amount of contrast medium injected is determined by the ease with which it flows into the disk, the reaction of the patient, the amount of pressure required to make the injection, and the degree of visualization as determined by fluoroscopy. Normal disks take 1 cc. or less with moderate pressure used during injection, although it is possible by using great pressure to inject as much as 2 cc. into a normal disk. The solution can be injected more readily into degenerated and ruptured disks and they require more solution for adequate visualization because it spreads readily throughout the areas of degenerated annulus and into any rupture that may be present. During the injection the pain experienced by the patient who has a herniated nucleus pulposus, is usually moderate to severe. Occasionally, it may be extremely severe, and in such patients the injection is temporarily stopped and then recommenced very slowly until visualization of the contrast medium is adequate.

I have injected 30 disks, of which 8 were believed to have herniations discographically. One of the 8 was believed to be a recurrent herniation following an operation for removal of a herniated disk, and a reoperation was not performed. The other 7 were operated on and a degeneration of the annulus with herniation was found in all. One patient had a ruptured disk which was found at operation, but not diagnosed by discography. Although the preliminary measurement in this patient showed that a 4-inch, 26-gage needle was required to reach the center of the disk, a 3½-inch needle was used. The injection of the contrast medium produced no symptoms, and although 2 cc. was injected, visualization was poor. The radiographs showed that most of the medium was in the epidural space, and the diagnosis was indefinite. This strengthens the belief that the injection should be into the center of the disk.

DISCUSSION

Gardner et al. (5) and Wise et al. (4) making no preliminary radiographs for the purpose of measuring the distance between the skin and the center of the disk, used a 3½-inch, 26-gage needle for the average patient. This may account for their relatively high incidence of the presence of contrast medium in the epidural space because a 3½-inch needle was found to be from 1/4 to 1/2 inch too short for the injection of the disk between the fourth and fifth lumbar vertebras in more than half of the adult patients examined here. Gardner (5) preferred to have the contrast medium injected into the posterior portion of the annulus fibrosus, rather than into the nucleus pulposus because the former is believed to be the area of prime interest. Other authorities (2, 6, 7) believed that the contrast solution should be injected into the nucleus pulposus because studies have shown that the solution will infiltrate throughout the nucleus and from it into all adjacent areas of degenerated annulus fibrosus. Furthermore, it has been shown that ruptures of the annulus occur obliquely and anteriorly as well as posteriorly (2, 7–9).

Neither Gardner et al. (5), Wise et al. (4), or Smith et al. (6) reported the making of oblique radiographs of the disks after injection. The important tangential visualization of a posterolateral herniation is not possible without oblique radiographs. The posterolateral approach has the advantage of avoiding puncture of the meninges. The use of a needle of excessive length obviates the need for measuring the patient to determine the length of needle required for reaching the center of the disk, but because of the slightly oblique entrance of the needle into the disk, and because fluoroscopy is not used, anteroposterior and lateral radiographs must be made in order to check the position of the needle, thus lengthening the procedure time.

(8) Cloward, R. B.: Anterior herniation of ruptured lumbar intervertebral disk; comments on diagnostic value of diskogram. Arch. Surg. 64: 457-463, Apr. 1952.

(9) Lindblom, K.: Discography of dissecting transosseous ruptures of intervertebral discs in the lumbar region. Acta radiol. 36: 12-16, July 1951.

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THE RIB AS A SECONDARY MISSILE[1]

WILLIAM J. GILLESBY, *Lieutenant Colonel, MC, U. S. A. R.* (2)

THE management of hemothorax has been well standardized and in war as well as in peacetime injuries can, in most patients, be adequately treated by repeated aspirations beginning 24 hours after the injury. Cuono (3) and Samson et al. (4) suggested waiting 48 hours or longer if the patient's condition permits that great a delay. Cuono believed that this delay permits natural sealing of bleeding points. If aspiration is inadequate, later decortication by thoracotomy is advocated by Blake et al. (5), Barden (6), Cameron et al. (7), and Forsee et al. (8).

The question of thoracotomy vs. aspiration is the subject of the present analysis. Blake et al. stated that thoracotomy was necessary in 11.7 percent of their patients. Cameron et al. stated that 3.1 percent of patients with stab wounds and 3.4 percent of those with gunshot wounds require decortication. Carr and Robbins (9) stated that between 15 and 20 percent of thoracic injuries observed during World War II clotted. The reason for the clot is the subject of much debate. Edwards and

(1) Reviewed in the Veterans Administration, and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

(2) Veterans Administration Hospital, Springfield, Mo., at time of writing; now with the Veterans Administration Hospital, Hines, Ill.

(3) Cuono, J. D.: Traumatic hemothorax. U. S. Nav. M. Bull. 43: 1107-1110, Dec. 1944.

(4) Samson, P. C.; Burford, T. H.; Brewer, L. A., III; and Burbank, B.: Management of war wounds of chest in base center; role of early pulmonary decortication. J. Thoracic Surg. 15: 1-30, Feb. 1946.

(5) Blake, H. A.; Wise, S. P., III; Taylor, V. S.; Kylar, S. L.; Major, R. C.; and Forsee, J. H.: Recent experiences in treatment of war wounds of chest. U. S. Armed Forces M. J. 2: 861-870, June 1951.

(6) Barden, S. P.: Important sequelae and complications of hemothorax resulting from penetrating wounds of pleural cavity. Am. J. Roentgenol. 59: 525-532, Apr. 1948.

(7) Cameron, D. A.; O'Rourke, P. V.; and Burt, C. W.: Management of penetrating and perforating wounds of chest in civilian practice. Am. J. Surg. 79: 361-368, Mar. 1950.

(8) Forsee, J. H.; Kylar, S. L.; and Blake, H. A.: Pulmonary function in traumatic hemothorax treated by decortication. J. Thorac. Surg. 22: 35-44, July 1951.

(9) Carr, D., and Robbins, S. G.: Streptokinase and antibiotics in the treatment of clotted hemothorax. Ann. Surg. 133: 853-866, June 1951.

Davies *(10)* stated that infection causes massive clotting, but Edwards *(11)* stated that there is doubt as to whether it is caused by a mild degree of infection or not Gardner *(12)* believed that the blood clots promptly in most patients but that the churning effect of the heart and lung defibrinates the clot so that the fibrin is deposited on the parietal and visceral pleura.

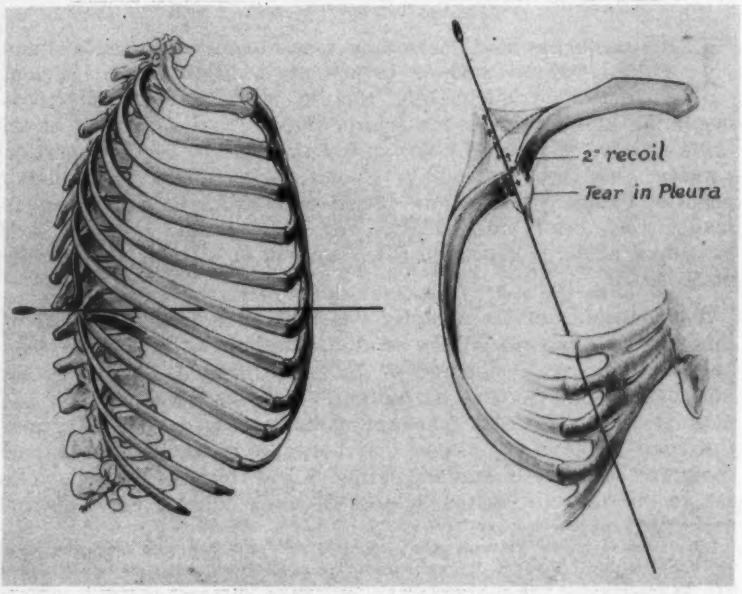

Figure 1. Diagram of missile entering the chest through an interspace and making its exit through a rib. It shows a 4-inch excursion outward and a 2-inch recoil at the point of fracture.

Hemothorax, acquired in combat, is usually the result of high-speed missiles and the course of the missile is important in forming a decision as to aspiration vs. open thoracotomy. The points presented here are the result of studying about 500 patients with hemothorax in the European Theater of Operations during World War II, as well as observation of a limited number seen in civil life. Bullets are generally kinder to tissue than shell fragments or other rough metallic bodies, but any missile

(10) Edwards, F. R., and Davies, H. M.: Traumatic hemothorax. Lancet 2: 673-675, Nov. 30, 1940.

(11) Edwards, A. T. (London): Traumatic haemothorax. Lancet 1: 97-99, Jan. 23, 1943.

(12) Gardner, C. E., Jr.: Symposium on aseptic surgical technic and its application to thoracic surgery; chest injuries; application of military experience to civilian practice. S. Clin. North America 26: 1082-1094, Oct. 1946.

striking a rib produces damage much greater than would ordinarily be expected because the velocity of the missile, striking a rib, is partially transmitted to the fractured rib and the rib becomes a secondary missile. The rib fragments are usually deflected from the path of the bullet.

The following observations were made:

1. If a missile enters the chest through an intercostal space and makes its exit through an intercostal space, aspiration will almost certainly be the only treatment required.

2. If a missile enters the chest through an intercostal space and strikes and fractures a rib on leaving the chest (fig. 1), aspiration will probably be sufficient. The rib being struck by the missile will spring outward. Its recoil will be of less force than its outward spring and the damage to the underlying lung will not be very serious.

3. If, on the other hand, the missile strikes and fractures a rib on entering the chest (fig. 2), aspiration will be sufficient in a small percentage of patients and open thoracotomy—resecting the fractured rib or ribs and removing bony spicules driven into the lung or other tissues—will be imperative in many. The damage to the underlying lung by indriven bone is

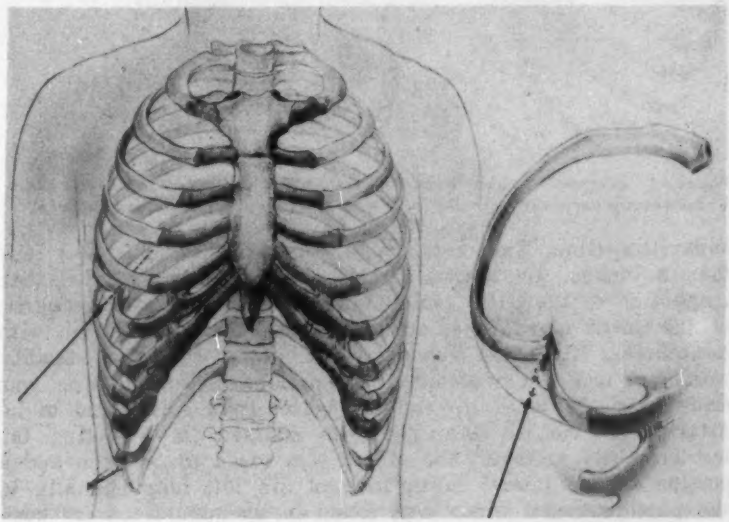

Figure 2. Diagram showing a missile entering the chest through a rib and producing a 4-inch incursion into the chest structures.

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much more serious than that done by the missile itself. Mediastinal wounds are not considered in this article.

4. If the missile strikes and fractures a rib both on entering and on leaving the chest, thoracotomy can rarely be avoided.

The amount of instantaneous motion of a rib fractured by a missile was not appreciated until the following case was seen.

CASE REPORT

A 26-year-old man was struck by a rifle bullet in the left lower lateral aspect of his chest (fig. 3). The wound of entrance was at the posterior axillary line and the wound of exit was at the

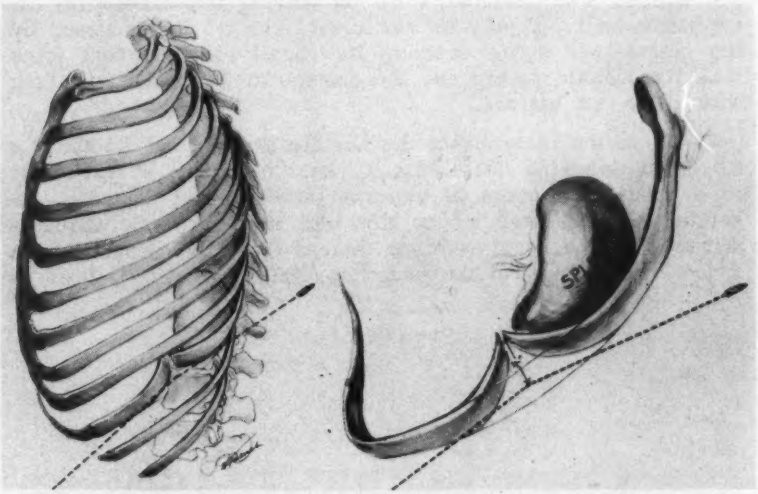

Figure 3. Diagram showing a missile producing a fracture of a rib which becomes a secondary missile lacerating the lung, diaphragm, and spleen.

midaxillary line. The distance between his 2 wounds was less than 3 inches. He seemed to be more severely wounded than inspection of the bullet holes would indicate. A roentgenogram of the chest revealed extensive fracture of the tenth rib with hemothorax. His chest was aspirated but his condition steadily worsened and a thoracotomy was performed. An incision was made over the tenth rib and the bullet tract was found to be external to the rib throughout its course. On resecting the fractured rib, however, the pleura was found to be open and a portion of the lateral lower lobe of his left lung normally in the phrenicocostal sinus was found in the pleural cavity completely severed from the rest of the lung. On cleaning the diaphragm of clots it was found to have a tear about 2 inches long. The diaphragm was further opened and the upper pole of

the spleen was found to be almost sheared off. The other abdominal organs were unharmed. A splenectomy was performed, the diaphragm closed, the lung sutured, and the chest closed with flutter valve seal of the drainage catheter. Recovery was uneventful.

DISCUSSION

The analysis of this case and the realization that the bullet fractured the tenth rib and the rib then became a secondary missile, springing into the chest, cutting the lung, diaphragm, and spleen to a depth of about 4 inches from the normal position of the rib was revealing. The amount of spring of a rib struck by a missile is from 4 to 6 inches (fig. 2), depending on the speed of the missile. An attempt has been made in recent years to demonstrate this experimentally without success. If serial x-ray exposures could be made at very high speed, or if a rib imbedded in gelatine could be prepared to show this graphically, the points could be further demonstrated.

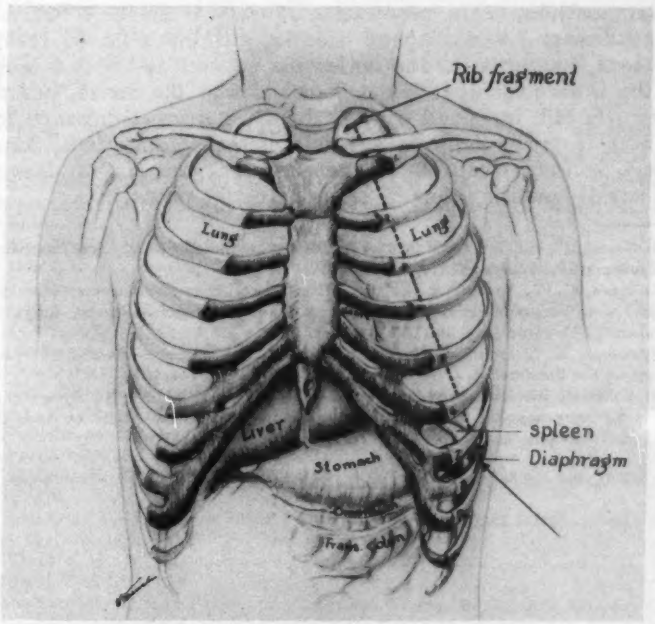

Figure 4. Diagram showing a missile striking a rib and a piece of rib fragment caroming off about 8 inches from and at almost a right angle to the path of the bullet.

Netter *(13)*, in his admirable collection of illustrations, brought out the fact that bullets carom off ribs, but the contention that rib fragments are deflected in equally bizarre fashion seems valid. One patient with a through-and-through bullet wound in the left lower portion of his chest was seen and a fragment of rib was found in the apex of his lung (fig. 4) about 8 inches and at almost a 90° angle from the path of the bullet.

The use of varidase was suggested by Carr and Robbins *(9)*, Read and Berry *(14)*, and Sherry et al. *(15)* and it was thought that these enzymes might reduce the necessity for thoracotomy in patients with hemothorax. Valle *(16)*, however, stated that this use of varidase has been abandoned in the Korean conflict.

SUMMARY

The mechanism of a high-speed missile striking a rib is similar to that that exists in billiards. The missile is deflected and the rib fragments carom off, depending on the angles involved. A high-speed missile, striking a rib on entering the chest, fractures it, and the fragments penetrate about 5 inches from their normal position. This penetration is only momentary but does severe damage. A high-speed missile, striking a rib on leaving the chest, fractures it. The deflection outward is about 4 inches and the inward recoil is about 2 inches from the normal position of the rib. Rib fragments may be found at a great distance from the path of the missile as a result of the carom effect. Thoracotomy is more likely to be necessary if the rib is damaged by a missile entering than by one leaving the chest.

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(13) Netter, F. H.: The Ciba Collection of Medical Illustrations. Ciba Pharmaceutical Products Co., Summitt, N. J., 1948. p. 55.

(14) Read, C. T., and Berry, F. B.: Utilization of streptokinase-streptodornase in patient with hemopneumothorax and patient with postpneumonectomy sanguineous coagulum. J. Thoracic Surg. 20: 384-392, Sept. 1950.

(15) Sherry, S.; Tillett, W. S.; and Read, C. T.: Use of streptokinase-streptodornase in treatment of hemothorax. J. Thoracic Surg. 20: 393-417, Sept. 1950.

(16) Valle, A. R.: War wounds of the chest: Experiences with 1,535 Korean casualties in an Army hospital overseas. Paper presented before the American Association for Thoracic Surgery, May 9, 1952, Dallas, Tex.

SICKLE CELL TRAIT AND FROSTBITE[1]

MATTHEW J. ELLENHORN, *Lieutenant, MC, U. S. A.*

DAVID WEINER, *Captain, MC, U. S. A.*

ABOUT 7 percent of normal Negroes exhibit the sickle cell trait (2–5), a mendelian dominant inherited tendency, which is easily demonstrated by exposing the red blood cells to a diminished oxygen environment or to the in vitro action of a number of reducing agents (6). In perhaps 1 of 40 Negroes with this trait, in vivo destruction of red blood cells will result in sickle cell anemia (2, 3, 7). A particularly important cause of symptoms in this disease is the occurrence of multiple vascular thrombotic occlusions resulting from the intravascular clumping of these abnormal cells (8). Little is known of the relationship of the sickle cell trait to the presence of agglutinogens of the AB and MN blood groups (9) and 2 investigations (10, 11) have shown that cold agglutinins are found in sickle cell anemia.

Among the factors postulated in the pathogenesis of frostbite are certain phenomena associated with slowing of the capillary blood flow: intravascular agglutination, clumping of red blood cells, exudation of serum, and possibly sludging of blood. Recent

(1) Army Medical Research Laboratory (Environmental Medicine Branch), Fort Knox, Ky.

(2) Diggs, L. W.; Ahmann, C. F.; and Bibb, J.: Incidence and significance of sickle cell trait. Ann. Int. Med. 7: 769-778, Dec. 1933.

(3) Lewis, J. H.: The Biology of the Negro. University of Chicago Press, Chicago, Ill., 1942. p. 235.

(4) Margolies, M. P.: Sickle cell anemia; composite study and survey. Medicine 30: 357-443, Dec. 1951.

(5) Sunderman, F. W., and Boerner, F.: Normal Values in Clinical Medicine. W. B. Saunders Co., Philadelphia, Pa., 1950. p. 43.

(6) Wintrobe, M.: Clinical Hematology. 3d edition. Lea & Febiger, Philadelphia, Pa., 1951. pp. 607-628.

(7) Sturgis, C. C.: Hematology. Charles C Thomas, Publisher, Springfield, Ill., 1948. p. 328.

(8) Diggs, L. W., and Ching, R. E.: Pathology of sickle cell anemia. South. M. J. 27: 839-845, Oct. 1934.

(9) Cardozo, W. W.: Immunologic studies of sickle cell anemia. Arch. Int. Med. 60: 623-633, Oct. 1937.

(10) McSweeney, J. E. J.; Mermann, A. C.; and Wagley, P. F.: Cold hemagglutinins in sickle cell anemia. Am. J. M. Sc. 214: 542-544, Nov. 1947.

(11) Young, L. E.: Clinical significance of cold hemagglutinins. Am. J. M. Sc. 211: 23-39, Jan. 1946.

evidence has accrued indicating the possibility that the presence of cold hemagglutinins may accelerate more complete red blood cell agglutination when a vascular area is exposed to low temperatures *(12)*.

Recent military operations in cold weather have shown that Negro troops are more susceptible than white troops to cold injury. Careless use of clothing and neglect of proper protection are probably the most important determinants in the pathogenesis of cold injury in these troops. Because intravascular agglutination may be an important factor in the evolution of cold injury, and because Negro troops have high cold hemagglutinin titers *(12)*, as well as the sickle cell trait, an attempt was made to study the role of sickling in the development of cold injury, its relation to cold agglutinins, and its relation to the main blood group agglutinogens.

METHODS

Subjects. Sickling tests were performed on Negro troops engaged in a cold weather military exercise during January and February 1952. Actual maneuvers occurred from 9 to 16 February 1952. A sickle cell preparation was studied on each of 221 Negro troops, selected at random, before maneuvers were begun. Similar preparations were studied on each of 66 Negro troops who manifested clinical frostbite during the maneuvers. A third group of 125 clinically normal Negro troops were tested for the sickle cell trait in the week following maneuvers.

Procedures. The sickle cell preparations were made by placing a drop of venous blood on a glass slide. The area was surrounded by petroleum jelly and covered by a coverglass, in effect sealing the red blood cells from any contact with the air. After 24 hours, during which they were kept at room temperature, the slides were examined under high-power and oil-immersion magnification. By this method, sickling, when present, was evident in almost all fields examined. All slides were read by one of us (MJE). All cold agglutinin tests were performed as in a previous study by Weiner *(12)*. The titers were read by one of us (MJE). All data on blood groups were obtained by direct questioning of troops and confirmed by examination of identification tags. Blood typing was not done as a rule. Statistical analyses were performed by the chi square method.

RESULTS

The results are shown in table 1. Because statistical analysis of the pre- and post-exposure groups showed no significant

(12) Weiner, D.: Role of cold hemagglutinins in frostbite. pp. 1-11. Report No. 67. AMRL Project No. 6-64-12-Z, 12 Oct. 1951.

difference in the incidence of sickling (p - 0.71), these 2 control groups were considered as homogeneous for purposes of comparison with the frostbite group. Comparison with the frostbite group with the combined control group also showed no significant difference (p - 0.9).

TABLE 1. *Sickling in frostbite and control subjects*

Group	Number	Sickling	Percent	p
Pre-exposure	221	24	10.9	0.71
Post-exposure	125	12	9.6	
Pre- and post-exposure	346	36	10.4	0.9
Frostbite	66	9	13.6	
Total	412	45	10.9	

Both sickle cell tests and blood group data were obtained on 40 patients with frostbite (table 2) and in 332 control subjects. The control group was further divided into 209 pre-exposure and 123 post-exposure subjects (table 3). There was no significant difference in blood group frequency between these latter groups. This was to be expected because blood groups are inherited.

The presence or absence of sickling in both the frostbite patients (table 2) and the control subjects (table 3) was found to have no statistically significant relation to their blood grouping. (For the frostbite patients p was 0.18; for the pre-exposure subjects, 0.15; and for the post-exposure subjects, 0.19.)

TABLE 2. *Sickling and blood groups in 40 patients with frostbite*

Blood group	Sickle cell trait				Total
	Positive		Negative		
	Number	Percent	Number	Percent	
O	5	20	20	80	25
A	3	33	6	6.7	9
B	1	25	3	75	4
AB	0	0	2	100	2
Total	9	22.5	31	77.5	40

TABLE 3. *Sickling and blood groups in control subjects*

Blood group	Sickle cell trait				Total
	Positive		Negative		
	Number	Percent	Number	Percent	
Pre-exposure					
O	15	13.4	97	86.6	112
A	2	3.8	50	96.2	52
B	6	15.8	32	84.2	38
AB	0	0.0	7	100.0	7
Total	23		186		209
Post-exposure					
O	10	12.0	73	88.0	83
A	1	4.2	23	95.8	24
B	1	7.7	12	92.3	13
AB	0	0.0	3	100.0	3
Total	12		111		123
Grand total	35		297		332

Table 4 summarizes the blood group frequency in the frostbite and control groups. No significant difference is apparent between the 2 groups. Table 5 summarizes the results of both sickle cell tests and cold agglutinin titers on 49 frostbite patients according to the presence of sickle cell trait. Within the group of frostbite patients analyzed the presence of the sickle cell trait was not associated with a significantly different amount of positive cold agglutinins. The number of patients available for analysis was

TABLE 4. *Blood groups in frostbite patients and control subjects*

Blood group	Subjects				Total	
	Control		Frostbite			
	Number	Percent	Number	Percent	Number	Percent
O	195	58.7	25	62.5	220	59.1
A	76	22.9	9	22.5	85	22.8
B	51	15.4	4	10.0	55	14.8
AB	10	3.0	2	5.0	12	3.2
Total	332		40		372	

small. Table 6 summarizes the results in the 209 pre-exposure subjects. Positive titers of cold agglutinins apparently were not present with greater frequency in the sickle cell positive group. Table 7 summarizes the results in the 123 post-exposure subjects. Again positive titers of cold agglutinins were not present with any greater frequency in the sickle cell positive group. The percent of subjects with positive agglutination (56.1) in this group may be significantly lower than that (69.4) in the pre-exposure group (p - 0.0145).

TABLE 5. *Sickle cell tests and cold agglutinin titers in frostbite patients*

Titers	Sickle cell trait				Total	
	Negative		Positive			
	Number	Percent	Number	Percent	Number	Percent
0	11		3		14	
1:2	17		5		22	
1:4	9				9	
1:8	2				2	
1:16						
1:32						
1:64	1				1	
1:128	1				1	
Total	41		8		49	
Positive agglutination	30	73.1	5	62.5	35	71.4
Negative agglutination	11	26.9	3	37.5	14	28.6

DISCUSSION

It is apparent from the data presented that the sickle cell trait is not a predisposing factor in the development of frostbite in Negro troops. No evidence has been presented in the literature to show that this trait has any causative relation to frostbite or cold, or that sickling "crises" in patients with sickle cell anemia are precipitated by low temperatures. The data presented substantiate the fact that exposure to cold does not increase sickling, again supporting the fact that the presence of the sickle cell trait is not influenced by external environmental factors during life, but is rather a constantly present heterozygous mendelian dominant characteristic. The data presented show little or no

TABLE 6. *Sickle cell tests and cold agglutinin titers in pre-exposure subjects*

Titers	Sickle cell trait				Total	
	Negative		Positive			
	Number	Percent	Number	Percent	Number	Percent
0	60		4		64	
1:2	44		6		50	
1:4	60		9		69	
1:8	10				10	
1:16	3		1		4	
1:32	8		2		10	
1:64	2				2	
Total	187		22		209	
Positive agglutination	127	68	18	81.8	145	69.4
Negative agglutination	60	32	4	18.2	64	30.6

TABLE 7. *Sickle cell tests and cold agglutinin titers in post-exposure subjects*

Titers	Sickle cell trait				Total	
	Negative		Positive			
	Number	Percent	Number	Percent	Number	Percent
0	49		5		54	
1:2	32		5		37	
1:4	24		2		26	
1:8	4				4	
1:16	1				1	
1:32						
1:64	1				1	
Total	111		12		123	
Positive agglutination	62	55.9	7	58.4	69	56.1
Negative agglutination	49	44.1	5	41.6	54	43.9

correlation between sickling and the presence of any particular blood group agglutinogen. Both sickling and blood groups are independent genetically acquired characteristics. Though cold agglutinins may be an environmentally acquired rather than an inherited trait *(13)*, little relationship to sickling was found in the Negroes studied.

CONCLUSIONS

In Negroes the presence of the sickle cell trait does *not* appear to be significantly related to the causation of frostbite. The appearance of the sickle cell trait apparently is *not* influenced by exposure to cold. Sickling has *no* apparent relationship to blood group agglutinogens in Negroes, or to cold agglutinins in control subjects and in patients with frostbite.

(13) Stats, D., and Wasserman, L. R.: Cold hemagglutination—interpretive review. Medicine 22: 363-424, Dec. 1943.

BOOK REVIEWS

Zinsser's Textbook of Bacteriology, by *David T. Smith,* M. D., Professor of Bacteriology and Associate Professor of Medicine, Duke University School of Medicine; *Norman F. Conant,* Ph. D., Professor of Mycology and Associate Professor of Bacteriology, Duke University School of Medicine; *Joseph W. Beard,* M. D., Professor of Surgery in Charge of Experimental Surgery, Duke University School of Medicine; *Hilda Pope,* Ph. D., Assistant Professor of Bacteriology, Duke University School of Medicine; *D. Gordon Sharp,* Ph. D., Assistant Professor of Biophysics in Experimental Surgery, Duke University School of Medicine; and *Mary M. Poston,* M. A., Instructor in Bacteriology, Duke University School of Medicine. 10th edition. 1,012 pages; illustrated. Appleton-Century-Crofts, Inc., New York, N. Y., publishers, 1952.

In the 10th edition of this widely known text the basic organization of material employed in previous editions has been retained. Sections are included on general biology of bacteria and general methods of bacteriology, infection and immunity, pathogenic micro-organisms (bacteria), the spirochetes, the pleuropneumonia group of organisms, Rickettsia and the rickettsioses, diseases caused by filtrable viruses, medical mycology, and technical methods. Sections on general biology and pathogenic bacteria have been condensed, while those on Rickettsia, infection and immunity, and the viruses have been greatly expanded to reflect recent emphasis on research in these fields.

Each chapter has been revised, many new and excellent illustrations are used, and a much more extensive bibliography is

appended to each chapter. Ease of reading and comprehension is facilitated by the use of boldface type for biologic concepts, fundamental descriptive terms, and key cultural reactions of organisms, while experimental data of a highly technical nature are denoted by small type. Infectious organisms are discussed from the standpoint, among others, of historical review, morphology and cultural behavior, identification, susceptibility to physical and chemical disinfection, mode of transmission, vectors, the clinical picture of their respective diseases, treatment, and prevention. Special attention has been given to the effect of the newer antibiotics on each organism.

An outstanding contribution to the text is the very complete discussion of the nature of viruses as a group, their physical and chemical properties, and methods of cultivation, purification, and titration. The laboratory technician will welcome the improved section on technical methods, with its complete array of standard biochemical tests, chick embryo culture methods, and serologic and immunologic technics; an extensive reference table on examination of specimens from patients and cadavers, listing suspected organisms, specific culture media and isolation technics, is included. The scope, completeness, and effective treatment of the material make this text an invaluable aid to the student of pathogenic microbiology, the clinical practitioner, and the public health worker.—*Capt. F. L. Davis, U. S. A. F. (MSC)*

Progress in Neurology and Psychiatry, Volume VII, An Annual Review, edited by *E. A. Spiegel*, M. D., Professor and Head of the Department of Experimental Neurology, Temple University School of Medicine, Philadelphia, Pa. 604 pages. Grune & Stratton, Inc., New York, N. Y., publishers, 1952. Price $10.

This book is a compilation of reports on recent psychiatric literature by 75 contributors. About 3,200 articles are cited as bibliographic references. The book is divided into sections on basic sciences, neurology, neurosurgery, and psychiatry. In view of the amount of material covered, it is remarkably complete and detailed and serves its purpose as a review. The section on basic sciences devotes much space to neurophysiology and pharmacology and presents these difficult subjects with unusual clarity. The section on neurology includes a new chapter on pediatric neurology. Otherwise it is similar to that in previous volumes. The neurosurgical section is the shortest but compares well with the others as a report of conclusions without detailed discussions of technics and experiments. The section on psychiatry is by far the longest section but successfully avoids verbosity. The book is well organized and indexed and will serve excellently as a reference when a brief review is desired.

—*Commander W. R. Griswold, MC, U. S. N.*

DOUBLE V VENTRAL SLIT CIRCUMCISION

J. HARWEDA WOOLFOLK, *Captain, U. S. A. F. R. (MC)* (1)

DAN HUEBERT, *Captain, U. S. A. F. R. (MC)* (2)

BECAUSE of the large number of military personnel who need or desire circumcision, a surgical procedure which would produce less pain postoperatively, reduce time lost from duty, and give a good cosmetic result was developed. Circumcision is indicated in adults for (1) phimosis; (2) paraphimosis with or without venereal and nonvenereal warts and ulcerated areas; (3) irritation resulting from the use of condoms and pressure of the prepuce during coitus; and (4) for cosmetic purpose. The two standard surgical procedures for circumcision are the dorsal slit and the guillotine technic. This report describes a new procedure, called the ventral slit, double V technic.

First the foreskin is retracted and double V incisions are made with a No. 15 scalpel blade. The distal V is made on the mucosa with the vertex beginning 2.5 cm. proximal to the corona and continuing distally on either side to a point 1 cm. from the corona. The two ends of the V are joined by a circular transverse incision extending around the penis (fig. 1). The proximal V incision is made in a similar manner, except that the vertex of the V is made from 1.5 to 2 cm. proximal to the mucocutaneous junction and continued distally on either side to a point 0.5 to 1.5 cm. from this junction. The two ends of the V incision are connected in a similar manner as the first V (fig. 1). The vertexes of the double V incisions are then joined by a vertical incision (fig. 2). The mucosa and the skin are dissected along the line of incision. Using sharp, curved scissors, careful dissection is necessary to prevent cutting of blood vessels under the subcutaneous tissue. It is important to dissect as closely as possible to the tissue being removed.

The proximal V is included in the dissected flap, but the distal V is left to be approximated in the space where the second V is absent. This tissue is left on the ventral surface to protect the

(1) U. S. Air Force Hospital, Bergstrom Air Force Base, Austin, Tex.
(2) U. S. Air Force Hospital, Rapid City Air Force Base, Weaver, S. Dak.

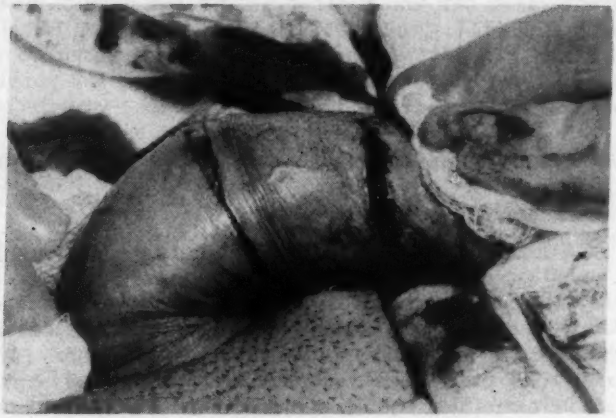

Figure 1. Photograph showing distal and proximal circular incisions.

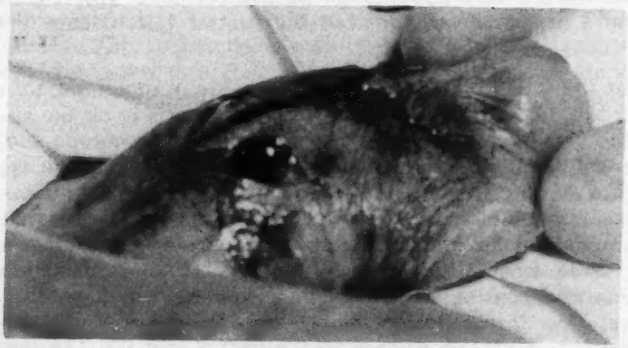

Figure 2. Photograph showing the vertical incision joining the two V incisions.

Figure 3. Photograph showing completed dissection.

frenum which is the most sensitive spot on the glans penis and is extremely painful postoperatively if it is damaged. The frenum is contained in the distal V as the dissection is completed (fig. 3). The proximal V is then approximated to the distal V and the subcutaneous tissue is joined with plain catgut sutures.

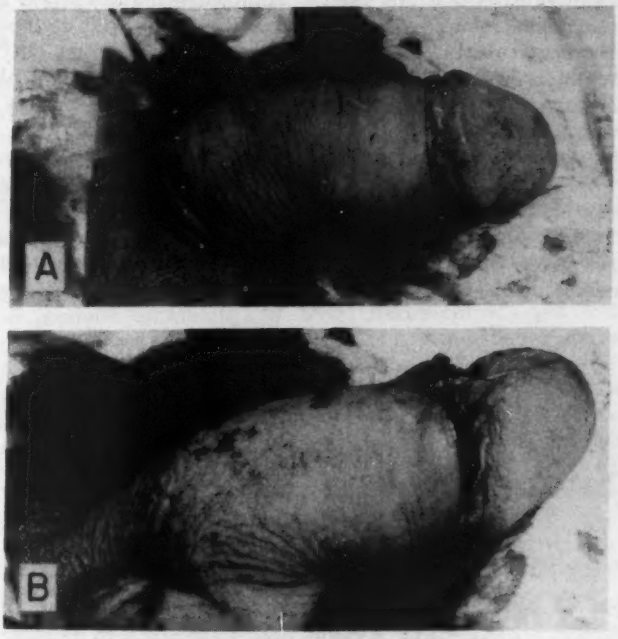

Figure 4. Photographs showing (A) *dorsal and* (B) *ventral surface of penis following completion of operation.*

The dorsal and ventral surfaces of the penis after the completion of the operation are shown in figure 4. The outer cutaneous edges of the wound are approximated to the mucosa with continuous No. 000 chromic catgut atraumatic subcuticular sutures beginning at the vertexes of one arm and continuing around to the other arm of the double V-shaped incision. The V-shaped incised edges are approximated with No. 0000 black silk interrupted sutures. The base of the penis is infiltrated with a local anesthetic to block the genitocrural nerve and branches of the ilioinguinal nerves. Minimal pain is experienced postoperatively and only 1 or 2 doses of demerol are required on the first day.

DISCUSSION

In performing the dorsal slit technic, a generous amount of skin must be left on the ventral side near the frenum to prevent

deformity of the penis during erection. The double V, ventral slit leaves sufficient mucosa on the frenum to assure that there will be no deformity during erection. Because of direct visualization, blood vessels are seldom cut when the redundant tissue is dissected.

The fine sutures that are used in approximating the edges of the incisions cause less postoperative pain because most of them are placed subcuticularly. Patients are discharged to light duty the first day after operation with small doses of codeine and phenobarbital to take at bedtime to prevent the pain that occurs from nocturnal erections.

SUMMARY

A new double V, ventral slit technic has been devised and successfully performed on 25 patients. It has the following advantages over other methods: (1) less postoperative pain. (2) less edema, and (3) quick return of the patients to normal activity.

Friends of the Armed Forces Medical Library

About 35 persons attended the dinner on 24 October 1952 in Washington, D. C., which brought into being the new organization called the Friends of the Armed Forces Medical Library. A constitution and bylaws were adopted. Salient provisions define the purpose of the Friends as being "to promote knowledge of, and interest in, the functions, resources, and services of the Armed Forces Medical Library, and to support programs for its development," and state that "membership in the Friends shall be open to anyone interested in the purpose of the Friends, on nomination by a member and approval by the Executive Committee." Meetings are to be held annually and dues were set at $2.00 a year. The Friends will sponsor the AFML News, previously underwritten by the Honorary Consultants, and will endeavor to offer general support to the Library as occasion may present.

It is hoped that many prospective members for the new organization will now come forward. All interested persons are advised to write to the Secretary-Treasurer, Friends of the Armed Forces Medical Library, Armed Forces Medical Library, Washington 25, D. C.

MERALGIA PARESTHETICA

WALTER J. FRIEDLANDER, *Captain, MC, U. S. A.* (1)

MERALGIA paresthetica is an entity which, though frequently misdiagnosed or unrecognized, is important in military medicine because it is not uncommon (2-5) and is capable of producing some degree of disability. Fourteen patients with this condition were seen in the outpatient clinic of this hospital from January 1951 to July 1952. Although this has been the commonest mononeuropathy seen in this clinic except for disease of the sciatic nerve, none of these patients had been diagnosed correctly. The referring diagnoses included: (1) neuritis of the second and third lumbar nerves, (2) sciatica, (3) probably psychogenic, and (4) pain, cause undetermined. Most of our patients with meralgia paresthetica had sufficient symptoms to produce disability. In 1895, Bernhardt (6) and Roth (7) independently, but almost simultaneously, described a neuropathy of the lateral femoral cutaneous nerve. Roth coined the term meralgia paresthetica from the Greek *meros* (thigh) and *algos* (pain). Since that time many reports of small series with, not infrequently, rather exotic causes have been published (8). In 1938 Ecker and Woltman (2) reported a series of 150 patients. Their report is used as a basis for the "classical" description of this disease.

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(1) U. S. Army Hospital, Fort Bragg, N. C., at time of writing; now with National Institute of Neurological Disease and Blindness, Bethesda, Md.

(2) Ecker, A. D., and Woltman, H. W.: Meralgia paraesthetica; report of 150 cases. J. A. M. A. 110: 1650-1652, May 14, 1938.

(3) King, B. B.: Meralgia paresthetica; report of 5 cases. Am. J. Surg. 52: 364-368, May 1941.

(4) Miller, J. W. (Seattle): Meralgia paraesthetica. Bull. Mason Clinic 1: 20-23, Mar. 1947.

(5) Schneck, J. M.: Meralgia paresthetica; age incidence. J. Nerv. & Ment. Dis. 105: 77-80, Jan. 1947.

(6) Bernhardt, M.: Ueber isolirt im Gebiete des N. cutaneus femoris externus vorkommende Parästhesien. Neurol. Centralbl. 14: 242-244, Mar. 15, 1895. Cited in Garrison, F. H.: Introduction to the History of Medicine. W. B. Saunders Co., Philadelphia, Pa., 1914.

(7) Roth, V. K.: Meralgia Paraesthetica, Physico-Medical Society of Moscow, Mar. 20-Apr. 1, 1895; brochure in German: Meralgia paraesthetica. S. Karger, Berlin, 1895. Cited in Garrison, F. H.: Introduction to the History of Medicine. W. B. Saunders Co., Philadelphia, Pa., 1914.

(8) Index Catalogue of Library of Surgeon General's Office: 2d. Ser. 10: 731-732, 1905; 3d. Ser. 7: 1192, 1928; 4th. Ser. 10: 842-843, 1948.

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DEFINITION

Meralgia paresthetica is a mononeuropathy involving the lateral femoral cutaneous nerve, usually unilaterally but occasionally bilaterally. Bernhardt was quite definite about the specificity of the nerve involvement, but Roth, in his initial report, cited several cases of "anterior meralgia paresthetica" (femoral nerve?) which he assumed had the same etiology as "external meralgia paresthetica." Confusion and inexactness apparently began here. The lateral femoral cutaneous nerve is, for clinical purposes, a purely sensory nerve with a quite specific distribution that covers the anterolateral and lateral aspects of the thigh. Reichert *(9)* pointed out that the nerve also carries sympathetic fibers. This explains any trophic or pilomotor changes that may be observed. Not restricting this disease to abnormalities of this single nerve has led to complicating a rather simple entity.

ETIOLOGY

Disease of the lateral femoral cutaneous nerve anywhere along its course will produce the symptoms of meralgia paresthetica, so a review of the course of this nerve is in order. The nerve arises from the second and third lumbar roots, passes beneath the psoas major muscle to emerge from the lateral portion of this muscle and then passes directly across the iliacus muscle. On the right side, this brings the nerve near the appendix and the cecum. It continues toward the anterior spine of the ilium. Near this point it turns sharply upward and passes beneath the short fibrous attachment of the internal abdominal oblique muscle and then under the inguinal ligament at which time it divides into 2 parts. The anterior or main branch remains beneath the superficial layer of the fascia only to pierce it eventually about 9 cm. below the inguinal ligament. It then supplies the outer aspect of the thigh down to the knee. The posterior or lesser branch courses posteriorly, becomes superficial almost at once, and supplies the skin from the greater trochanter downward *(10)*.

As an aid in determining the cause of the neuropathy, the nerve may be divided into three parts: (1) the spinal cord and the roots of the second and third lumbar nerves, (2) the intra-abdominal portion, and (3) the distal portion which passes over the ilium, under the inguinal ligament and into the fascia. Although it is difficult to visualize disease of the cord or the roots of the second and third lumbar nerves producing only sensory loss over the distribution of the lateral femoral cutaneous nerve without loss elsewhere over the distribution of these nerves, Ecker and

(9) Reichert, F. L.: Meralgia paraesthetica, form of causalgia relieved by interruption of sympathetic fibers. S. Clin. North America 13: 1443-1449, Dec. 1933.

(10) Lee, F. C.: Meralgia paraesthetica. Internat. Clin. 1: 210-299, Mar. 1936.

Woltman *(2)* reported 5 patients in their series with the lesion in this location. Two of these were ultimately diagnosed as multiple sclerosis; one as a residuum of radiculitis; one as osteitis of the second and third lumbar vertebras secondary to a perinephric abscess; and one as acute bilateral meralgia paresthetica which developed shortly after a spinal anesthesia. The last-mentioned patient also had intermittent spasms of the tensor fasciae latae muscle which, using our definition of meralgia paresthetica, would exclude it as an example of this disease.

Because of the course of the nerve in the abdominal cavity, disease in the abdomen in juxtaposition to the nerve can produce the signs and symptoms of meralgia paresthetica. The cecum and the appendix lie near the right lateral femoral cutaneous nerve, hence carcinoma or abscess of these organs may be the cause of a neuropathy. Miller *(4)* reported a patient with a left ureteral calculus and a left-sided meralgia paresthetica. Dislodgement and passage of this stone afforded immediate and complete relief of his neuropathy. Tumors of the uterus or pelvic inflammatory disease have been reported as causes of meralgia paresthetica. Pregnancy has occasionally been associated with this particular neuropathy although the cause here might well be pressure on the nerve in its distal rather than on its intra-abdominal part.

Injury to the nerve in its third or distal portion probably accounts for most cases. This injury might be direct and obvious such as that incurred by one of our patients who suffered from a shell fragment wound of the left hip. It might be due to pressure without any other injury, as in the army officer (reported by Bernhardt) whose disease was caused by the pressure of his scabbard. Trusses, belts, and corsets have been implicated at times. One of the most common causes in this group is obesity. Roth observed that meralgia paresthetica was most common in those who were overweight. The average weight of 136 patients in Ecker and Woltman's series was 175 pounds which they believed was "greatly in excess of the average weight of middle-aged men and women." They stated that their figures were "not statistical proof that obesity is causally related to meralgia (but) the association of the two conditions is suggested." They also pointed out that it may not be the weight of the patient but rather its distribution that is important. The panniculus may exert tension on the nerve and produce undue demands on the inguinal ligament and the fascia of the thigh for support.

About one-third of the patients in our series developed symptoms soon after coming into the Army. This group had no apparent cause, not even obesity. Another patient of ours, not included in our series, had the onset of his symptoms shortly after induction into the Navy. Although he has grown more obese

since his release from the service, his symptoms have decreased. These cases suggest that the physical rigors of military service (long marches, calisthenics, et cetera) may put unusual stress on the tensor fasciae latae and hence be one of the precipitating causes of this illness.

Toxins, metabolic disorders, and infections were so rare in Ecker and Woltman's series that they believed these played no appreciable part in the cause of the disease in their patients. Other authors *(10, 11)* listed such things as alcohol, morphine, lead, diabetes, gout, herpes zoster, typhoid fever, grippe, rheumatic fever, and syphilis (independent of a localized gumma) as possible causes.

SIGNS AND SYMPTOMS

If a rigid definition is used for this disease there should only be a sensory defect over the distribution of the lateral femoral cutaneous nerve. In our series the distribution was usually oval in shape and the involvement was unilateral. Not infrequently the sensory disturbance starts in a small area in the lower lateral aspect of the thigh and progressively becomes larger. The signs are limited to a hypalgesia, hypesthesia, and hypothermia over part or the entire distribution of the nerve. The patients usually complain of numbness or burning. Most of our patients complained of "pins and needles," burning, or shooting pains over the distribution of the nerve when they stood for any length of time or walked for a long distance. These difficulties were most bothersome when the soldier had to stand at attention at parade, or in some even if it was only as long as standing for retreat. Marching the length of a parade ground was sufficient to produce paresthesias in several patients, though others noted their symptoms only if required to hike several miles. Roth *(12)* reported a 10-year-old girl whose pain was made worse when she lay down.

The motivation of the soldier is important in evaluating the disability produced by this illness. One officer had sharp, shooting pains whether at rest or in motion and was unable to continue his work which was limited to light duty in an office. When he was offered surgical treatment with the assurance of relief and return to full duty, he refused. On the other hand, a well-motivated officer from a combat unit, whose meralgia paresthetica followed a fracture of the pelvis due to a parachute jump, could not be adequately followed because he placed so little emphasis on his symptoms even though he presented the typical findings of this disease the one time he was seen. This difficulty in evaluating the disability produced by this illness is not limited to the military service but is also seen in civilian practice *(11)*.

(11) Huddleson, J. H.: Meralgia paresthetica. Am. J. M. Sc. 175: 823-831, June 1928.
(12) Roth, P. B.: Meralgia paresthetica. Brit. M. J. 2: 536, Nov. 1916.

TREATMENT

Most of the patients in our series were adequately treated by explaining to them the nature of the illness and its limited symptomatology. This latter factor is particularly important for many of our patients were more impressed with the progressive character of the illness than with the symptoms themselves. Not infrequently it was necessary to excuse the soldier from long marches or prolonged standing. Three patients were given 120 micrograms of vitamin B_{12} intramuscularly each day for 2 weeks. This was based on the empiric use of this drug in other neuritides with reported good results *(13, 14)*. In our hands, this therapy was useless. One patient, who had developed his symptoms shortly after coming into the service, who had an adequate military record despite his meralgia paresthetica, and who was to be discharged from the Army in several months, had symptoms severe enough to warrant surgical resection of the lateral femoral cutaneous nerve. He was followed for only a short time, but when last seen he found the anesthesia following operation not very bothersome and the relief from the sharp pains most gratifying. The consensus of others *(10, 15)* is that if the symptoms are severe, resection of the nerve is the treatment of choice. This is a relatively simple operation because of the superficial location of the nerve as it emerges beneath the inguinal ligament. The nerve can usually be easily blocked with procaine and hence a trial of this before resection is indicated so that the patient can judge whether he prefers paresthesias or analgesia. This can also be used as a prognostic guide to the efficacy of the surgical treatment. Heat, rest, and a reducing diet have been advised. Treatment of any underlying cause is of the utmost importance.

PROGNOSIS

We have not had the opportunity to follow any of the patients in our series for a sufficient time to reach any conclusions about their ultimate fate. The longest duration of symptoms in a patient when first seen in this clinic was 10 years; the shortest was a few weeks; the mode was about 1 year. In Ecker and Woltman's series the duration of symptoms ranged from 35 years to less than 1 month. They concluded that "when the symptoms have lasted less than 2 years, the chances are 2 to 1 that they will disappear spontaneously within another 2 years."

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(13) Fields, W. S., and Hoff, H. E.: Relief of pain in trigeminal neuralgia by crystalline vitamin B_{12}. Neurology 2: 131-139, Mar.-Apr. 1952.

(14) Use of vitamin B_{12} in neurology. (Foreign Letters, Paris) J. A. M. A. 148: 667, Feb. 23, 1952.

(15) Brain, W. R.: Diseases of the Nervous System. Oxford Medical Publications. 3d edition. Oxford University Press, New York, N. Y., 1947.

SUMMARY

Meralgia paresthetica is not an uncommon disease, but it is usually overlooked or misdiagnosed. It is due to some abnormality of the lateral femoral cutaneous nerve. It frequently causes some degree of disability although this must be evaluated in the light of the motivation of the patient. Conservative treatment consisting of rest, limitation of duty, and explanation of the illness usually suffices. Resection of the nerve may at times be necessary.

BOOK REVIEW

The Old Egyptian Medical Papyri, *by Chauncey D. Leake,* Vice-President, Medical Branch, University of Texas, Galveston. Logan Clendening Lectures on the History and Philosophy of Medicine, Second Series. 108 pages. University of Kansas Press, Lawrence, Kans., publisher, 1952. Price $2.

This book presents the lectures delivered by the author in the second series of the Logan Clendening Lectures on the History and Philosophy of Medicine. Aimed frankly at the general reader interested in the development of medicine, this slender volume is informational rather than critical in its approach. For example, the author briefly describes the 8 known medical papyri and goes into some detail in discussing the physical aspects of the Hearst Papyrus. To this reviewer, the most interesting sections were those dealing with old Egyptian weights and measures and ancient Egyptian therapeutics. The appendix furnishes 260 prescriptions listed in the Hearst Medical Papyrus, a collection of "various recipes" offered as a "guide for treatment" after the physician had examined the patient and reached a diagnosis.

Dr. Leake's thesis, following the reasoning of Doctors Owsei Temkin, Hermann Ranke, and Hermann Grapow, is that, contrary to what one might expect, ancient Egyptian medicine "seems to have been empirically rational." He notes, however, that the "copying of recitals and abjurations into the formularies" indicates that then, even as today, there was a dichotomy between the "rational, empirical, and essentially scientific approach to disease by professional physicians' and the "anxiously confused, irrational, and superstitious attitude of the people generally."—*M. M. Link, Ph. D., Historian, U. S. A. F.*

BIGEMINY DUE TO AUTONOMIC NERVOUS SYSTEM IMBALANCE

SEYMOUR L. ALTERMAN, *Captain, U. S. A. F. (MC)* (1)

MORRIS M. DICK, *M. D.* (2)

A patient who developed bigeminal rhythm when assuming the recumbent position was observed. Although the literature fails to reveal reports of similar cases, it is probably not a rare occurrence, particularly in young neurasthenic patients.

Sesenbach and Buci (3) discussed the occurrence of persistent ventricular bigeminal rhythm in apparently normal hearts caused by overexertion, excitement, emotional stress, and fatigue. Excessive use of coffee, tea, alcohol, tobacco, or digitalis may produce extrasystoles. They did not correlate postural changes as a cause of bigeminy in their series of patients. In a review of 12,000 ECG's, Parsonnet et al. (4) noted the various patterns which exhibited bigeminal rhythms, but did not include bigeminal rhythm induced by the recumbent position. White (5) noted that premature beats could be elicited by pressure on the carotid artery or by sudden pain. Friedberg (6) believed that premature beats arise when an ectopic focus in the heart becomes so irritable that the rate of its impulses exceeds that of the sinus node. He noted that stimulation of the vagus and sympathetic nerves induced extrasystoles in dogs. Weiss and English (7) discussed the emotional factors responsible for the production of premature beats.

Wendkos (8) pointed out the profound influence of autonomic imbalance on the human ECG. He was able to demonstrate un-

(1) Dobbins Air Force Base, Marietta, Ga.

(2) Veterans Administration Hospital, Coral Gables, Fla.

(3) Sesenbach, W., and Buci, R. M.: Persistent ventricular bigeminal rhythm in apparently normal hearts. Am. J. M. Sc. 211: 332-335, 1946.

(4) Parsonnet, A. E.; Miller, R.; Bernstein, A.; and Klosk, E.: Bigeminy, electrocardiographic study of bigeminal rhythms. Am. Heart J. 31: 74-92, Jan. 1946.

(5) White, P. D.: Heart Disease. Macmillan Co., New York, N. Y., 1946. pp. 852-863.

(6) Friedberg, C. K.: Diseases of the Heart. W. B. Saunders Co., Philadelphia, Pa., 1949. p. 248.

(7) Weiss, E., and English, O. S.: Psychosomatic Medicine. W. B. Saunders Co., Philadelphia, Pa., 1943.

(8) Wendkos, M. H.: Influence of autonomic imbalance on human electrocardiogram; unstable T waves in precordial leads from emotionally unstable persons without organic heart disease. Am. Heart J. 28: 549-567, Nov. 1944.

stable T waves in precordial leads from emotionally unstable persons without heart disease. The patients in his series showed clinical evidence of neurocirculatory asthenia. He believed that the distortion of the T wave sometimes was caused by hypervagotonia as he could exaggerate the changes with sympatholytic drugs. In addition, changes in the T waves were brought about by assumption of the recumbent position. The changes produced by recumbency could be abolished by the preliminary use of atropine, a parasympatholytic drug. No direct relationship to cardiac rate was demonstrated. Wendkos believed that assuming the upright position caused increased sympathetic tone, reflexly induced, and that this was not caused by changes in the position of the heart. He also noted similar effects in sympathicotonic persons in whom T-wave and S-T segment changes were caused by adrenergic preponderance. Atropine and ergot derivatives verified this by causing either intensification of the abnormality or return to normal. He believed that aberrations could be caused by vagotonic influences which were exaggerated by a sympatholytic drug, and abolished by a parasympatholytic drug or by the assumption of the upright position. He also stated that an altered T wave, which is normalized by a sympatholytic drug and exaggerated by a sympathomimetic drug, can be related to increased sympathetic tone.

Electrocardiographic changes can be produced by autonomic nervous system influences, although, in his observations, Wendkos stressed S-T wave changes primarily. Other electrocardiographic changes, such as premature ventricular contractions or bigeminy, may also occur in autonomic nervous system imbalance as demonstrated in the following case report.

CASE REPORT

A 20-year-old man was referred by his draft board for evaluation of multiple complaints. A tentative diagnosis of brucellosis had been made. He complained of vague aches and pains in his knees and legs, shortness of breath manifested by sighing respiration, profuse axillary perspiration, cold clammy hands, and palpitations in the chest.

Physical examination revealed a slender, somewhat nervous man who had (1) moist palms; (2) normal cardiac rhythm on standing; (3) bigeminal rhythm when reclining; (4) a normal blood pressure with no evidence of marked variation in different body positions; and (5) normal cardiac findings on fluoroscopy and roentgenography.

Electrocardiographic studies were made in an attempt to show the relationship of the influence of the autonomic nervous system in the development of bigeminy. Figure 1 is an ECG showing

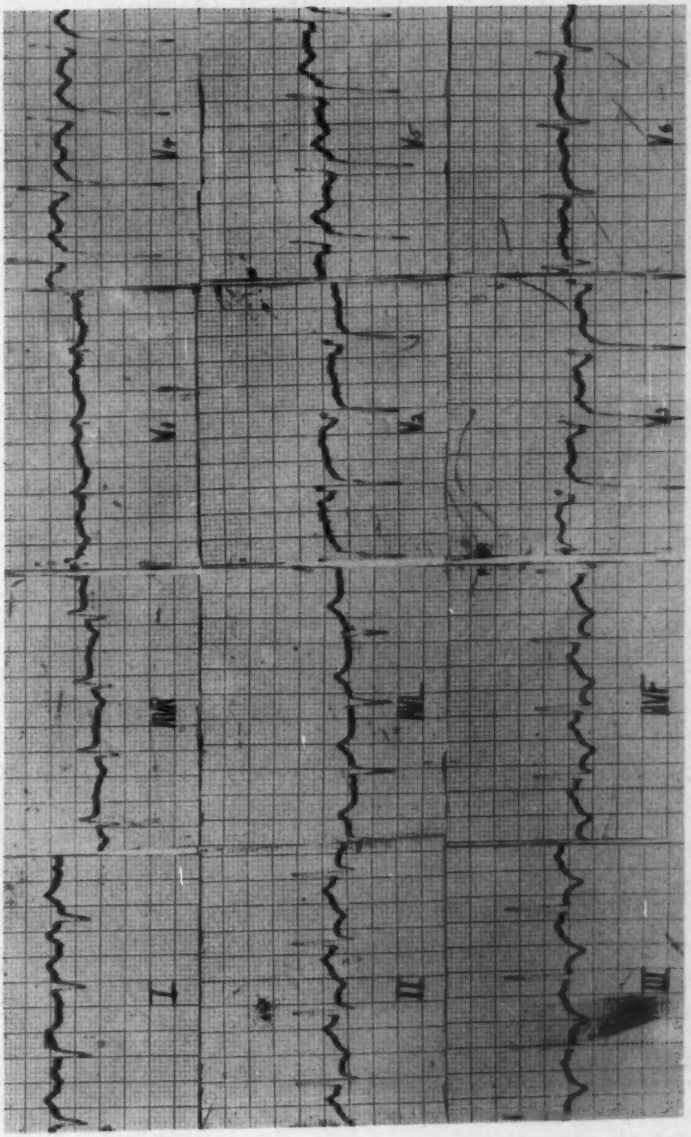

Figure 1. Apparently normal tracings obtained while patient was sitting upright.

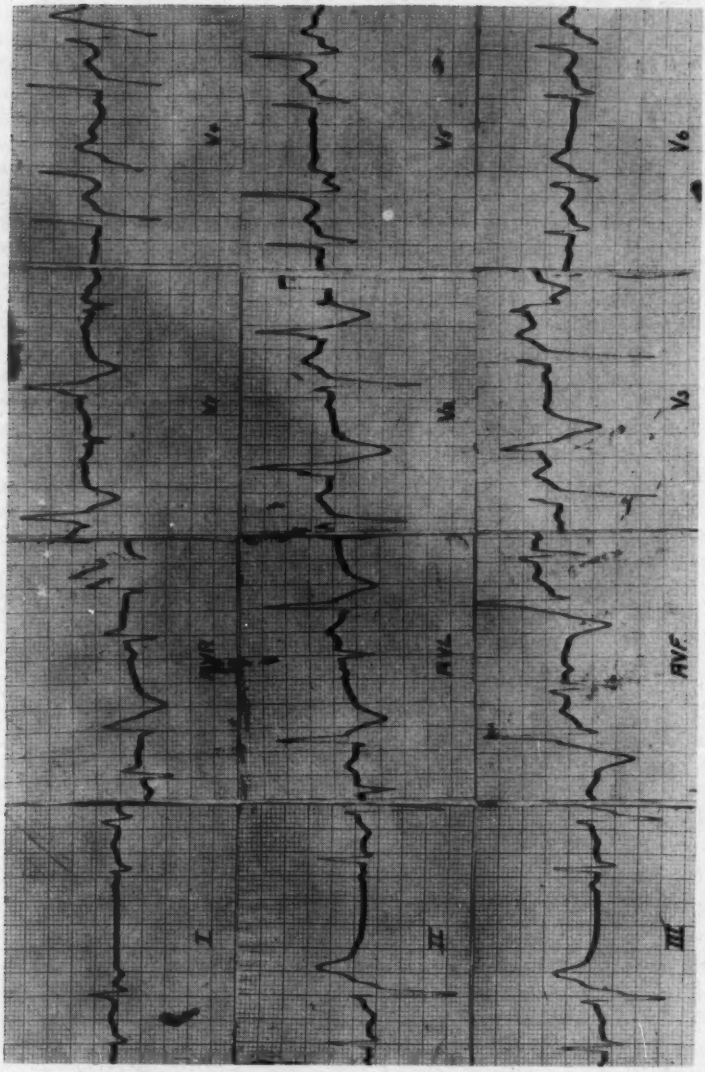

Figure 2. Tracings show bigeminal rhythm in all leads following the patient's assumption of the recumbent position.

regular sinus rhythm while the patient was sitting upright. Figure 2 shows development of bigeminal rhythm with the ectopic focus arising from the left ventricle when he assumed the recumbent position. This arrhythmia was reproduced in this patient whenever he assumed this position. It persisted for 5 minutes or longer then the rhythm would revert spontaneously to regular sinus rhythm (fig. 3). Vagotonic influences were responsible for

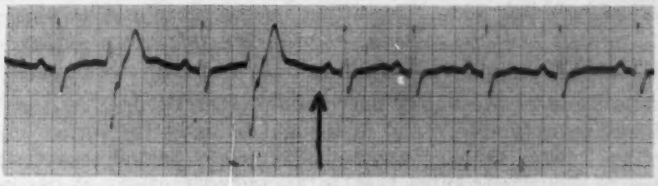

Figure 3. Lead II tracing showing reversion of bigeminal rhythm to regular sinus rhythm after 5 minutes of recumbency.

the development of premature ventricular contractions and bigeminal rhythm as shown in figure 4. The arrhythmia was reproduced by pressure on the carotid sinus after the patient developed regular sinus rhythm following prolonged recumbency. This observation was substantiated when the patient was given 1.2 mg. of atropine subcutaneously and allowed to assume the recumbent position. While under the effect of atropine he failed to develop bigeminy, even after carotid sinus pressure but as the effect of the atropine wore off in about 45 minutes it was again possible to induce premature ventricular contractions and bigeminy. This demonstrates the predominant role of vagotonic influences in the development of bigeminal rhythm in this patient.

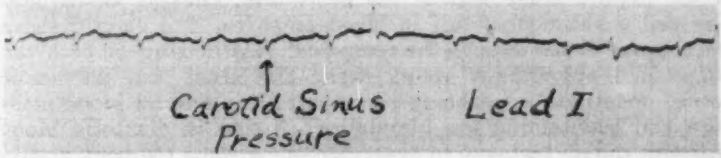

Figure 4. Tracing showing bigeminy following carotid sinus stimulation.

The inversion of the T waves in leads II, III, and aV_F (fig. 1) when the patient was in the upright position were probably due to sympathicotonia *(8)*. This again demonstrates the autonomic nervous system imbalance present in this patient.

DISCUSSION

Although much experimental and clinical study has been accomplished, there is only scant knowledge regarding the actual regulation of the heart. We have learned that cardiac behavior is

subject to nervous, chemical, and mechanical influences. The medulla receives impulses via the vagus nerve and is influenced by the numerous cardiac reflexes. Pressure on the eyeballs, stimulation of the fifth cranial nerve, emotional stress, et cetera, all produce afferent reflexes in the cardiac center. The vagus nerves are cardio-inhibitory and act through the deep and superficial cardiac plexuses. From the latter, impulses traverse the auricular myocardium, ganglion cells, postganglionic fibers, and the sino-auricular node. The effect on the auricle is to slow the beat, weaken the contraction, shorten the refractory period, and raise the threshold of excitation.

Stimulation of the vagus nerve retards the cardiac rate mainly by lengthening diastole and is thought to exert its action on the auricular musculature. Best and Taylor *(9)* stated that the decrease of the ventricular rate is an indirect effect of auricular slowing or depression of conduction in the auriculoventricular connections. Stimulation of the left vagus causes ventricular slowing by depressing A-V conduction and blocking auricular impulses with little effect on the auricular rate.

The spinal accelerator cells located in the lateral horns of the upper thoracic segments of the spinal cord connect with the inferior, middle, and superior cervical ganglia and when stimulated increase the heart rate via the sympathetic nervous system. Stimulation of the accelerator fibers located in the superior, middle, and inferior cardiac nerves causes a rise in blood pressure and increase both in the heart rate and the force of contraction. The accelerator nerves act directly on ventricular muscle. Thus we have two antagonistic and reciprocal nervous mechanisms controlling cardiac rate and behavior. Pressure on or stimulation of the carotid sinus retards the heart rate, causing vasodilatation and a subsequent fall in blood pressure. The carotid sinus mechanism works through the reciprocal relationship of both the vagus and sympathetic reflex arcs. The sinus and the aortic nerves constitute a mechanism for controlling arterial blood pressure and maintaining the circulation. A rise in diastolic blood pressure and an increase in heart rate occur when the body changes from a recumbent to the sitting position.

The electrocardiographic evidence shown can provoke speculation on the mechanisms for the production of premature ventricular contractions caused apparently by a change in body position. There is a shift of blood from the upper half of the body to the splanchnic area and lower extremities when we assume the erect position. The reverse occurs in recumbency. This affects

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(9) Best, C. H., and Taylor, N. B.: Physiological Basis of Medical Practice. Williams & Wilkins Co., Baltimore, Md., 1950.

the aortic nerves and carotid sinus reflex arcs, and various sympathetic and parasympathetic reflex impulses are activated to maintain adequate circulation. Gammon and Bronk *(10)* recorded the impulses in the peripheral ends of the splanchnic nerves and their branches in cats. They noted that the carotid sinus and aortic nerve endings lie within the adventitia of large vessels in which the degree of distention is determined by the blood pressure. They believed that the pacinian corpuscles in the mesentery discharge afferent impulses when changes in circulation occur. The sudden rush of blood from the splanchnic area may cause afferent impulses to arise from the pacinian corpuscles and bring about changes in cardiac rate and rhythm.

It is believed that premature beats arise when an ectopic focus in the heart becomes dominant or perhaps when the normal impulse arising from the sino-auricular node is inhibited. If this is so, we must assume that changes in position cause parasympathetic impulses via the vagus nerve to inhibit the normal cardiac impulse arising in the S-A node allowing an irritable focus in the ventricle to initiate a premature contraction. Although this sounds plausible, it must be remembered that the auricular muscle does not receive innervation from the vagi. For this reason we can only theorize on the actual nervous mechanism for the production of these premature beats. We must also take into account the fact that these cardiographic changes occurred in neurasthenic patients. Does this imply greater autonomic imbalance in these people? Undoubtedly so in some neurasthenics with vagotonia and other forms of sympatheticotonia. Wendkos' work demonstrated this fairly conclusively. In our patient with bigeminal rhythm, atropine prevented the production of ectopic beats even after carotid sinus stimulation, which convincingly implicated the vagus nerve.

(10) Gammon, G. D., and Bronk, D. W.: Discharge of impulses from pacinian corpuscles in mesentery and its relation to vascular changes. Am. J. Physiol. 114: 77-84, Dec. 1935.

BOOK REVIEW

Nursing Care of Communicable Diseases, Prophylactic Technics for the Prevention and the Control of Disease, by *Mary Elizabeth Pillsbury,* R. N., M. A.; and *Elizabeth Johanna Sachs,* R. N., B. S., M. Litt. Science Adviser and Editor: *Jean Broadhurst.* Contributors: *Jessie Wright* and *Jane Taylor Torrance.* 7th edition. 843 pages; illustrated. J. B. Lippincott Co., Philadelphia, Pa., publishers, 1952.

This is a well-written book, arranged in four parts, covering all phases of communicable diseases. Part I is an introduction to

communicable diseases and covers causal organisms, disease control (which includes carriers, relative legislation, government and private agencies in public health), immunity, and the numerous other introductory factors relative to communicable diseases. The excellent and interesting discussion on antibiotics can be easily understood by students. The presentation of nursing technics is clear, concise, unbiased, and includes disinfection, sterilization, prophylactic and miscellaneous technics employed in homes and hospitals.

Part I is a basic, essential preliminary to Part II which is an alphabetical arrangement of the communicable diseases. Each disease is thoroughly covered by definition, history, occurrence, and medical aspect (etiology, source of infection, route of transfer, incubation period, period of communicability, laboratory diagnosis, clinical picture, prognosis, complications and sequelas, treatment, measures of control, nursing aspect, and special isolation and disinfection recommendations). To facilitate notations relative to new discoveries, reports, et cetera, blank pages have been inserted following the discussion of each disease.

Supplementary materials (Part IV) are covered in 5 sections and are useful in both teaching and learning. The section on disinfection procedures (a supplement to chapters in Part I) includes methods and materials to be used on various articles. For the teacher, as well as for the avid student, there is a list of general references, audio-visual aids, and situation questions.

As a whole, the charts, graphs, and photographs are excellent. There are a few, however, which this reviewer considers undesirable. Figures 40-44 and 54-57 show the use of long-sleeved gowns. It is the opinion of this reviewer, as a result of experience, that it is practically impossible to remove such a gown without contaminating the inside of the long sleeves, especially those that have snug-fitting cuffs. Other illustrations considered undesirable are figures 44 and 50 showing scrub sinks that are of the operating room type, a style with a breadth that invites contamination by body contact with the outer edge of the sink. Sinks used in isolation units should be of such design that it would not be impossible to keep from touching the sink while scrubbing. Figures 39 and 47 show the desirable style. This, however, is a minor criticism of an excellent textbook for teaching and one that every nurse, particularly if engaged in medical or communicable-disease nursing, would find to be a handy reference book.—*Lt. Comdr. G. E. Dvorak, NC, U. S. N.*

TRAUMATIC ANEURYSM OF THE INNOMINATE ARTERY[1]

EVERETT H. DICKINSON, *Captain, MC, U. S. N.*
R. MAURICE HOOD, *Lieutenant, MC, U. S. N.*
FRANK C. SPENCER, *Lieutenant, junior grade, MC, U. S. N. R.*

INJURIES to the innominate artery and other great vessels of the mediastinum are not infrequent. The majority of persons so injured die immediately or within a few minutes from exsanguination or pressure phenomenon. A review of the available literature reveals that ligation of the innominate artery is an infrequently performed procedure. Lindskog (2) stated that 107 ligations of the innominate artery had been reported. Of these, 75 were performed for spontaneous aneurysms, 11 for recent injuries of the innominate artery or one of its branches, and 1 for control of remote hemorrhage. Twenty ligations were performed for traumatic aneurysm of the innominate artery or one of its branches. Twelve of these involved the subclavian; 5, the innominate; and 3, the carotid artery.

Shumacker (3) reported that ligations for traumatic aneurysm of the innominate artery had been attempted on 37 patients of whom 19 survived. Rundle (4) reported the first successful triple ligation and also a successful triple ligation with excision of the aneurysmal sac. Langley (5) reported a patient who had a through-and-through bullet perforation of the bifurcation of his innominate artery, in whom bleeding had spontaneously ceased, probably as a result of shock. He was explored a few hours after injury because of the appearance of signs of cerebral damage from carotid occlusion. He died a few hours after the operation, probably because of carotid ligation. Brock (6) reported an unfortunate example of failure to open the aneurysmal sac. His

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(1) U. S. Naval Hospital, Oakland, Calif.

(2) Lindskog, G. E.: Surgery of innominate artery. New England J. Med. 235: 71-76, July 18, 1946.

(3) Shumacker, H. B., Jr.: Surgical cure of innominate aneurysm; report of case with comments on applicability of surgical measures. Surgery 22: 729-789, Nov. 1947.

(4) Rundle, F.: Aneurysm of innominate artery treated by surgery; report of 3 cases and records of 22 cases collected from literature. Brit. J. Surg. 25: 172-190, July 1937.

(5) Langley, G. F.: Gunshot wound of innominate artery. Brit. M. J. 2: 711-712, Dec. 4, 1943.

(6) Brock, R. C.: Aneurysm of innominate artery; report of case treated surgically. Guy's Hosp. Rep. 90: 180-195, 1940-1941.

patient survived ligation of the innominate and carotid arteries only to succumb to a secondary hemorrhage from rupture of the aneurysm because of a patent internal mammary artery.

Other writers (*7–14*) have reported patients who were treated surgically.

CASE REPORT

On 16 September 1951 in Korea, a 19-year-old marine was struck by shell fragments and incurred multiple small wounds of his neck and the anterior aspect of his chest. Immediate shock resulted and he responded promptly to the administration of plasma and blood. A small swelling was noted at the base of the right side of his neck. He was transferred the same day to the U. S. Naval Hospital, Yokosuka, Japan. A thoracentesis revealed a right hemothorax which was treated by daily needle aspiration. The swelling in the neck gradually increased in size. Fifteen days after injury, the right upper extremity became completely paralyzed.

The patient was transferred to this hospital 1 month after injury. A hard mass measuring 15 by 15 cm. filled the right side of his neck (fig. 1). No pulsation was palpable in the mass and no bruit was heard. His trachea deviated markedly to the left (fig. 2). His left carotid pulse was normal but the mass prevented palpation of his right carotid pulse. There was a total loss of motor and sensory response in his right upper extremity. No arterial pulsation could be felt in his right arm. A Horner's syndrome was present on his right side and he was moderately hoarse. His general physical condition was good. A roentgenogram of the chest revealed a large mass filling the apex of his right hemithorax and the superior mediastinum, producing marked deviation of his trachea to the left. There was a diffuse density over the right side of his chest from a residual hemothorax.

(*7*) Wheeler, W. I. de C.: Ligature of innominate artery for right subclavian aneurysm; end-result. Brit. M. J. 2: 422-423, Oct. 10, 1942.

(*8*) Elkin, D. C.: Arteriovenous aneurysm; approach to innominate vessels. J. A. M. A. 129: 26-27, Sept. 1, 1945.

(*9*) Holman, E.: Surgery of large arteries with report of case of ligation of innominate artery for varicose aneurism of subclavian vessels. Ann. Surg. 85: 173-184, Feb. 1927.

(*10*) Trent, J. C.: Ligation of innominate artery for innominate aneurysm using rubber bands; report of case. Surg., Gynec. & Obst. 82: 463-470, Apr. 1946.

(*11*) Daniel, R. A., Jr.: Aneurysm of subclavian artery in infant; report of case. J. Thoracic Surg. 14: 487-489, Dec. 1945.

(*12*) Williams, M. H.: Accidental incision of aneurysm of left common carotid artery; treatment by thoracocervical excision with recovery. Ann. Surg. 135: 267-277, Feb. 1952.

(*13*) Bigger, I. A.: Treatment of traumatic aneurysms and arteriovenous fistulas. Arch. Surg. 49: 170-179, Sept. 1944.

(*14*) Greenough, J.: Operations on innominate artery; report of successful ligation. Arch. Surg. 19: 1484-1544, Dec. 1929.

He stated that the mass had decreased slightly in size in the preceding few days. It was considered that he had a traumatic arterial injury near the bifurcation of his innominate artery. Surgical exploration was advised, but on the afternoon of the third hospital day he developed a marked stridor in conjunction with slight cyanosis. A pulse rate of from 120 to 130 beats per minute also developed. Preparations for an immediate operation were made.

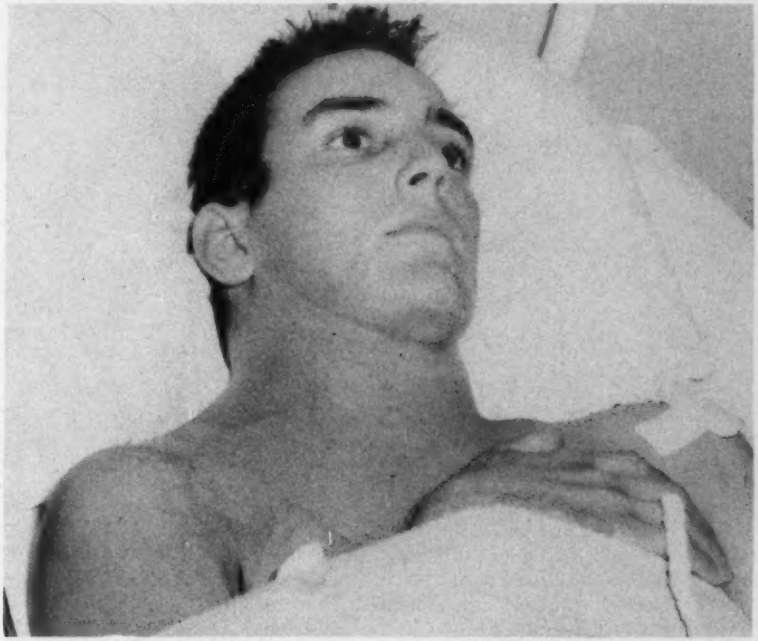

Figure 1. Photograph, taken about 1 hour prior to operation, showing the aneurysmal mass filling the right side of the neck and supraclavicular area.

The patient was deeply cyanotic by the time he reached the surgery. Immediate intubation of the trachea without anesthesia resulted in prompt improvement. A curved incision was made extending over the medial two-thirds of his clavicle and down over his sternum to the level of the fourth interspace (fig. 3). The medial two-thirds of the clavicle was resected subperiosteally, and the periosteal bed incised posteriorly. The tissues were markedly infiltrated with blood. Superficial exploration with the finger resulted in severe arterial bleeding which was controlled by packing.

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The lower end of the skin incision was then extended laterally over the right third interspace. The pleural cavity was entered through the third interspace and the first 4 costal cartilages were severed near the sternum. About 1,000 cc. of blood were evacuated from the pleural space. The mediastinal pleura was intact, but the aneurysmal mass filled the superior

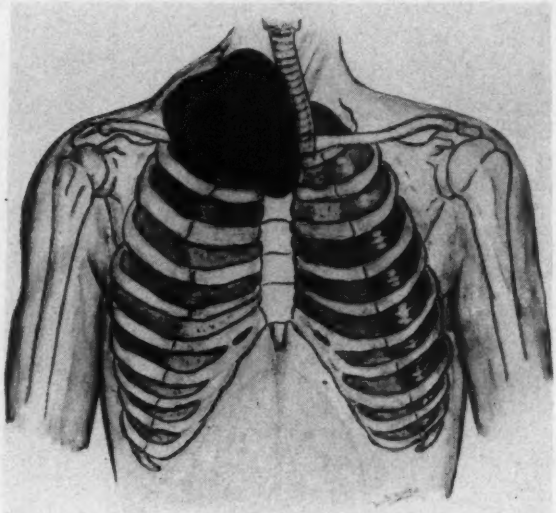


Figure 2. Drawing showing the relative size of the aneurysm and the tracheal deviation.

mediastinum and protruded into the apex of the pleural space for about 5 cm. The mediastinum was entered and the superior vena cava retracted anteriorly in an attempt to isolate the innominate artery at its origin from the aortic arch. Dense fibrosis from shell fragments prevented separation of the mediastinal structures, although the arch of the aorta could be palpated.

The vessels were partially compressed digitally at the aortic arch and the sternum was split with the Lebsche sternum shears to the third interspace and transected into the right third interspace. As the split sternum was retracted, severe arterial hemorrhage again occurred. This was controlled with difficulty by packing. At this point the patient had an estimated blood loss of 5,000 or 6,000 cc. Blood was being given under pressure in 1 arm and 2 leg veins; 6,000 cc. of blood had been infused, and the patient's general condition remained good.

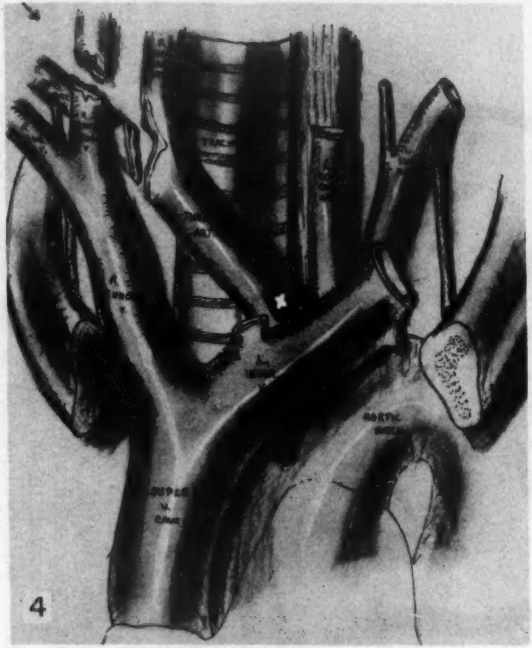
The major difficulty was the inability to isolate the innominate artery proximal to the point of injury. The left third interspace

was opened and the left pleural space was entered, but the innominate artery could not be isolated. The only remaining possibility was to open the aneurysm directly and secure the bleeding points. With the vessels at the aortic arch partially compressed digitally, the aneurysm was widely opened. About 900 cc. of old clotted blood were evacuated in the presence of extremely severe arterial hemorrhage and the innominate artery was secured by digital compression. From 1,500 to 2,000 cc. of additional blood loss were sustained before the artery was secured.

The subclavian artery had been completely transected just distal to its origin, the open end of the vessel being clearly visible in the base of the wound. An extensive rent was also present in the common carotid artery at its origin (fig. 4). The common carotid, subclavian, and innominate arteries were doubly ligated individually with No. 20 cotton sutures. The right subclavian and internal jugular veins had been injured and were

Figure 3. Drawing showing topographic anatomy of the chest and the extent of the excision.

also ligated (fig. 5). The exact nature of the injuries to the veins could not be clearly ascertained. The hematoma was evacuated of as much old blood as possible, leaving a cavity which had an approximate volume of 500 cc. in the superior mediastinum and base of the neck. The chest wall was reconstructed with sutures of No. 32 steel wire. The remainder of the wound was closed with interrupted No. 20 and No. 40 cotton sutures. Intercostal tubes were left in the third interspace in the anterior axillary line and in the ninth interspace in the posterior axillary line. A Penrose drain was placed in the hematoma. A tracheotomy was performed at the termination of the procedure.



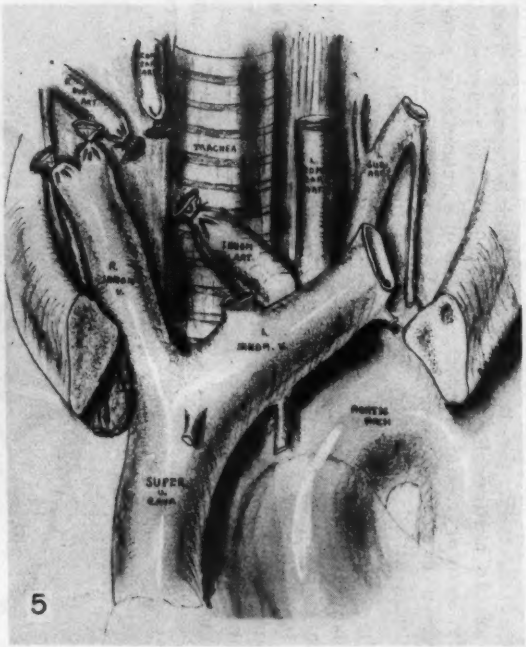


Figure 4. Drawing of the vascular anatomy showing the extent of the vascular injury at the bifurcation of the innominate artery and to the right internal jugular vein. The pathway (arrow) and final resting place (X) of the shell fragment are shown. Figure 5. Drawing showing the ligations performed in correcting the vascular defect.

The patient's general condition remained surprisingly good throughout the operation, which lasted 6 hours. A total of 11,500 cc. of blood was given. The patient's blood type was AB, and only through the resourcefulness of the blood bank personnel was the patient's survival possible. At the end of the operation his entire right upper extremity was cool and cyanotic. The upper skin flap in his right third interspace had a similar appearance at the time of closure. His condition was otherwise satisfactory.

Postoperatively, he showed no sign of cerebral damage. His right arm became moderately swollen and was treated by elevation, elastic bandages, alcohol given intravenously, and intramuscular injections of papaverine. The coldness and cyanosis of his hand apparently originated from venous stasis as a result of ligating the subclavian vein. All symptoms had subsided by the fifth postoperative day. No arterial pulsation was palpable in his arm but his skin remained warm and of good color. He became moderately jaundiced by the second postoperative day, probably as a result of a mild hemolytic reaction from multiple transfusions totaling 11,500 cc. His urinary output was satisfactory, and the jaundice slowly subsided.

The skin incision over his sternum partially separated with the subsequent drainage of large amounts of purulent material, exposing the underlying third costal cartilage. There was no evidence of secondary hemorrhage from the ligated vessels. His chest wall rapidly became rigid. The exposed cartilage was excised on the twenty-first postoperative day, after which the wound rapidly healed. The tracheotomy tube was removed on the tenth postoperative day. The patient had a mild, persistent hoarseness although his vocal cords appeared normal. The Horner's syndrome on his right side persisted. At the time of his transfer to a hospital near his home on 21 December there had been no return of nerve function in his right upper extremity although there was no evidence of vascular insufficiency.

DISCUSSION

Traumatic injuries of large intrathoracic vessels are rarely seen because they are rapidly fatal. The reported survivors had only small wounds of entry which prevented rapid exsanguination. The presence of a small skin wound, however, does not rule out a serious vascular injury.

This case illustrates the pressure effects of an expanding aneurysm on adjacent nerves. Although the brachial plexus had been paralyzed for only 2 weeks, the injury was complete. In the 2-month period during which he was under our observation there was no evidence of nerve regeneration. Posttraumatic

aneurysms that begin to expand follow a uniform course of increasing pressure effects until spontaneous rupture occurs, and any evidence of expansion should be an immediate indication for surgical intervention.

No case of gangrene of the hand following ligation of the innominate artery has been reported, thus demonstrating the efficiency of the collateral circulation about the shoulder girdle. Only 2 patients with gangrene of the hand following ligation of the subclavian artery while undergoing the Blalock-Taussig subclavian-pulmonary artery anastomosis for tetralogy of Fallot have been reported *(15)*.

The incidence of cerebral symptoms following ligation of the common carotid artery in young people has been reported to be from 5 to 20 percent. The subclavian artery had been completely divided in our patient and the common carotid artery was torn and probably occluded completely from the pressure of the aneurysm. It is probable that the triple ligation performed altered the cerebral blood supply very little from that of the preoperative state.

The cyanosis and coolness of the right arm in the immediate postoperative period was alarming, but subsided promptly in the first few postoperative days. Similar changes are seen in patients with so-called "effort thrombosis" of the axillary vein, but have not been described in the operative cases reported in the literature when the innominate vein was divided.

Inability to isolate the innominate artery because of mediastinal fibrosis was the main cause of difficulty in this patient. The extension of the aneurysm into the mediastinum to the level of the arch made attempts to isolate the vessel more hazardous. A more thorough use of the transverse incision with wide exposure of the left pleural cavity might have given better access to the origin of the innominate artery at the arch of the aorta.

The absence of instability of the chest wall illustrates the ability of the soft tissues to compensate for marked bony defects in it.

The large amount of blood given with the maintenance of relatively good circulatory dynamics throughout the operation demonstrates the ability of the body to tolerate massive hemorrhage when the blood is promptly replaced. Intra-arterial transfusion might have given an additional factor of safety because decreased coronary blood flow and myocardial failure

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(15) Webb, W. R., and Burford, T. H.: Gangrene of arm following use of subclavian artery in a pulmonosystemic (Blalock) anastomosis. J. Thoracic Surg. 23: 199-204, Feb. 1952.

from massive hemorrhage may be refractory to the intravenous replacement of blood.

SUMMARY

In a patient with a traumatic aneurysm of the innominate artery, who was cured by triple ligation, resection of the clavicle and splitting of the sternum provided the necessary exposure. This had been obstructed by the immense size of the aneurysm and by the presence of fibrosis around the great vessels. Massive rapid transfusion, using multiple veins, permitted successful conclusion of the procedure with its attendant blood loss.

BOOK REVIEWS

Disorders in Perception, With Particular Reference to the Phenomena of Extinction and Displacement, by *Morris B. Bender,* M. D., Professor of Clinical Neurology, New York University College of Medicine; Director, Neurological Service, Bellevue Hospital; Neurologist, Mt. Sinai Hospital, New York City. 109 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1952. Price $3.

Appreciation of the sensations of two simultaneous stimulations was first described by Oppenheim in 1885. This book discusses the present-day wider application of the responses to two stimuli. In the past few years this method of examination has become better understood and it is now being used more and more by the clinician. Certain brain lesions, which cannot be localized by single stimulations, may be lateralized and/or localized to a definite area by using this test. It may be of value, too, in determining on which side an arteriogram should be done in certain cases.

Simultaneous stimulations may be by various means. The two main significant responses discussed in the book are extinction of one of the two simultaneously applied stimuli as the patient is (or becomes) unaware of one of them, and displacement of sensation of one stimulus so that it is described as being at a spot other than where the stimulation is actually applied. (Throughout the book there is a repeated effort to convince the reader that the author's term of 'extinction' is more appropriate than Dr. Critchley's term of 'inattention.') Some of the most interesting work described in the book is in perimetric studies, as for example in the author's patient in whom a hemianopsia was discovered by using two images but no hemianopsia was found when only one image was used.

There is no single area in the brain where the duality of the stimulation is registered; and, indeed, the author describes the testings as revealing (1) lesions in the thalmus, internal capsule, parietal lobe, temporal lobe, and occipital lobe, (2) diffuse brain disease, and (3) degenerative diseases involving the corpus callosum. He also points out that this type of testing has been of value in delimiting the level of a spinal cord lesion.

A chapter is devoted to the various factors which may influence the patient's response to simultaneous stimuli such as (1) intensity of the stimulus, (2) the constant predominance of sensation in the face over more caudad sensations, and (3) the influence of background—both the environmental and the patient's own intrinsically organized pattern of reaction.

This book is recommended to neurologists and neurosurgeons. The portion devoted to early identification of certain hemianopsias should be of value to the ophthalmologist.

—*Lt. Comdr. G. Clark, MC, U. S. N.*

Pictorial Handbook of Fracture Treatment, by *Edward L. Compere,* M. D., F. A. C. S., Associate Professor of Surgery, Northwestern University Medical School; Chairman, Departments of Orthopedic Surgery, Wesley Memorial and Children's Memorial Hospitals; Consultant in Orthopedics, Chicago Memorial and Henrotin Hospitals; Medical Adviser and Director, Rehabilitation Center of the Liberty Mutual Insurance Company, Chicago, and *Sam W. Banks,* M. D., F. A. C. S., Associate Professor of Surgery, Northwestern University Medical School; Attending Orthopedic Surgeon, Chicago Memorial, Woodlawn and Hines Veterans Hospitals, Chicago. Revised with the assistance of *Clinton L. Compere,* M. D., F. A. C. S., Assistant Professor of Surgery, Northwestern University Medical School; Attending Orthopedic Surgeon, Wesley Memorial Hospital; Consultant in Orthopedic Surgery, Augustana Hospital; Senior Consultant, Veterans Administration, Chicago. 424 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1952. Price $6.50.

This is a concise, lucid, and precise treatise dealing with the most recent and finest methods and procedures in the treatment of fractures. As an example of the excellent directives in treatment the following is quoted from the chapter on the hand: "Every injured finger should be immobilized until torn structures have had time to repair (and) the fracture has healed * * *. Immobilization should be restricted to the injured finger or fingers with the joint kept in flexion whenever possible. Active exercise of the uninjured fingers is imperative." The step-by-step outline in technic of internal fixation or the closed reduction of fractures correlated with descriptive drawings, roentgenograms, and diagrams make this an invaluable book for the medical officer and an indispensable aid for the general practitioner, resident, intern, and medical student.

—*Capt. J. L. Schwartz, MC, U. S. N. (Ret.)*

CARCINOMA OF THE LIVER WITH BONE METASTASES RESEMBLING MULTIPLE MYELOMA

JOHN C. BATES, *Major, MC, U. S. A. (1)*

PRIMARY carcinoma of the liver is relatively rare. Charache *(2)*, in a survey of the records of 31 institutions, found 808 (0.5 percent) instances of primary carcinoma of the liver in 159,762 autopsies. Hoyne and Kernohan *(3)* found 0.227 percent in European and American peoples and a slightly higher incidence in Asiatics. Other authors give similar low percentages.

In Charache's study he added to his own 808 instances, 317 from the literature, making a total of 1,125. He reported only 18 instances of skeletal metastasis. Willis *(4)*, in describing 15 patients with carcinoma of the liver, reported 2 with metastasis to bone. Spatt and Grayzel *(5)* reported 17 patients with carcinoma of the liver of whom 22 percent showed skeletal metastases. Mallory *(6)*, discussing a patient with bone involvement, commented that carcinoma of the liver is one of the types of carcinoma that metastasizes with great regularity to bone. These reports occur in the literature after the study made by Charache. It is suggested that skeletal metastases may occur more frequently than Charache indicated and that the finding of such metastases may be accounted for by (1) the heightened awareness of the possibility of skeletal metastasis in this disease and (2) the increasing use of roentgenographic examination in diagnosis.

Carcinoma of the liver is difficult to diagnose clinically and most cases are diagnosed either very late or at autopsy. It is not surprising then that a skeletal or other type of metastasis may be the presenting finding. Willis pointed out "that skeletal or other metastasis may clinically simulate primary disease or be the first

(1) Gorgas Hospital, Ancon, C. Z.

(2) Charache, H.: Primary carcinoma of liver; report of case and review of literature. Am. J. Surg. 43: 96-105, Jan. 1939.

(3) Hoyne, R. M., and Kernohan, J. W.: Primary carcinoma of liver; study of 31 cases. Arch. Int. Med. 79: 532-554, May 1947.

(4) Willis, R. A.: Pathology of Tumours. C. V. Mosby Co., St. Louis, Mo., 1948. pp. 430-437.

(5) Spatt, S. D., and Grayzel, D. M.: Primary carcinoma of liver. Am. J. Med. 5: 570-573, Oct. 1948.

(6) Mallory, T. B.: Case Records of Massachusetts General Hospital. New England J. Med. 219: 133-135, 1938.

sign of illness" as in his paraplegic patient who had bony metastasis. Moon's *(7)* patient presented himself with a pathologic fracture of a femur. Bolker et al. *(8)* also reported a patient with a pathologic fracture of the left femur as the initial complaint. Mallory's patient had few clinical findings in addition to an area thought to be a metastasis in the ulna. The patient reported here also shows the diagnostic difficulties because osseous metastasis at first clouded the diagnosis.

CASE REPORT

A 44-year-old Panamanian was admitted on 15 May 1950 complaining of pain in the left side of his chest of 6 weeks' duration. The pain had gradually become more severe and was aggravated by deep breathing and by assuming certain positions. The patient thought he had had a fever on several occasions, but was vague as to the specific time of day it occurred or its duration. There were no night sweats or cough, but he thought he had lost some weight.

The patient appeared to be chronically ill. There was a questionable pleural friction rub over the base of his left lung. His hemoglobin was 12.24; his erythrocyte count was 3,680,000; and his leukocyte count was 4,600 with 62 percent neutrophils, 33 percent lymphocytes, 3 percent eosinophils, and 2 percent basophils. These hematologic findings are considered average for native Panamanians. His sedimentation rate was 33. A roentgenogram of the chest revealed a pleural reaction in the left base with associated blunting of the left costophrenic sulcus. Multiple circumscribed areas of rarefaction were present in the lower ribs bilaterally (fig. 1). Roentgenograms of the skull revealed multiple punched-out areas of rarefaction. There was destruction of both the inner and outer tables of the skull and there was no reaction about these areas (figs. 2 and 3). A tentative diagnosis of multiple myeloma was made on the basis of these findings.

Fourteen subsequent blood studies showed findings essentially the same as those reported. Six examinations for Bence-Jones proteins in the urine were negative. The total serum protein on 18 May was 8.14 mg. per 100 cc.; on 20 May it was 8.83 mg. per 100 cc. (albumin 3.70 and globulin 5.13); on 16 June it was 8.98 mg. per 100 cc. (albumin 3.60 and globulin 5.38). Acid phosphatase was 2.5 King-Armstrong units and alkaline phosphatase was 26.6 units. Sternal marrow examination showed no evidence to confirm the diagnosis of multiple myeloma.

---

(7) Moon, V. H.: Primary carcinoma of liver with metastasis to bone. Arch. Path. 8: 938-943, Dec. 1929.

(8) Bolker, H.; Jacobi, M.; and Koven, M. T.: Primary carcinoma of liver with bone metastasis. Ann. Int. Med. 10: 1212-1221, Feb. 1937.

The patient was presented to the tumor board, and here grave doubt was cast on the diagnosis of multiple myeloma. Further marrow studies and a biopsy of a rib were recommended. The patient refused these procedures. Because of a current interest in urethane, the patient was given a course of this drug. He improved

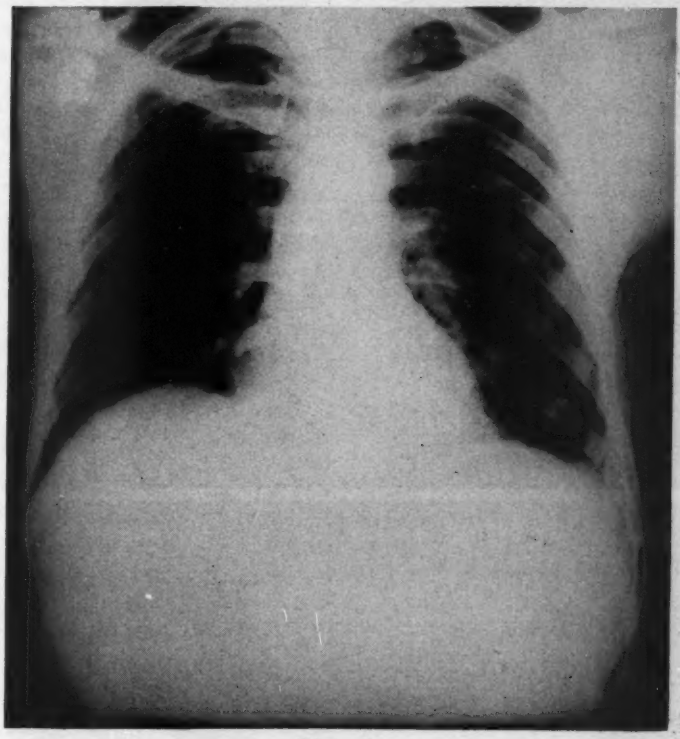

Figure 1.

clinically and became very eager to return to work. A definite diagnosis could not be made and because of the patient's marked clinical improvement he was discharged on 30 June with the advice to return periodically to the outpatient clinic for observation.

He was readmitted on 9 August complaining of nausea and vomiting of 24 hours' duration. He stated that the vomitus was streaked with bright red blood. He had no abdominal pain and up to the time of admission had not had any blood in the stool. He thought that he had been having some fever and increased fatigability for about 3 weeks. He appeared to be emaciated. His

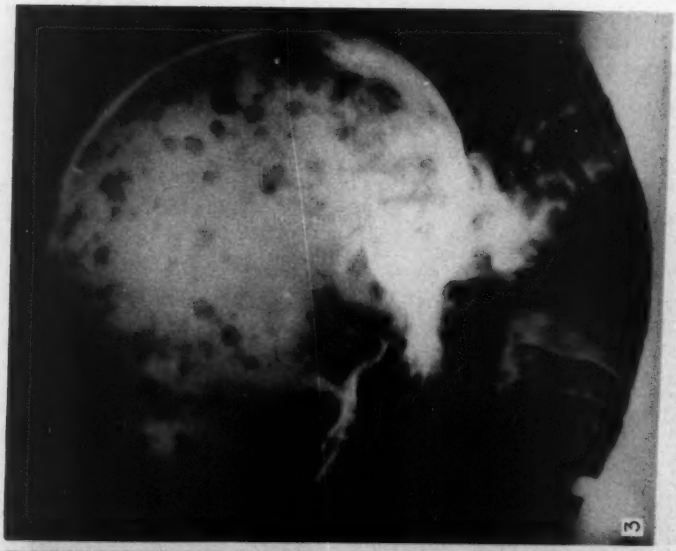

Figure 3.

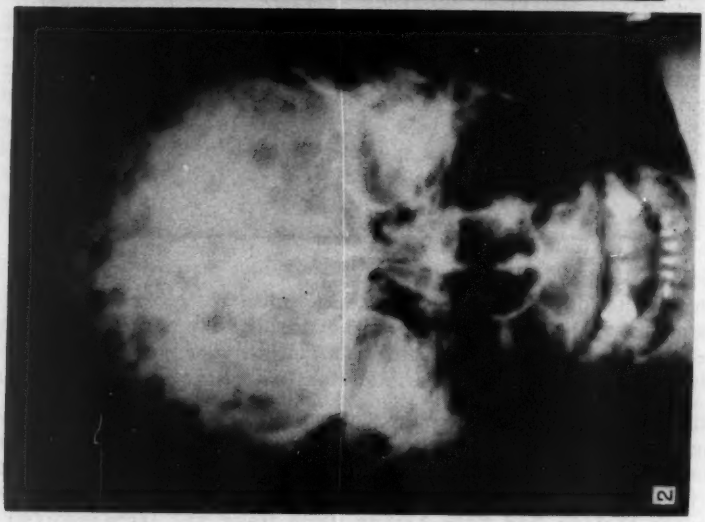

Figure 2.

abdomen appeared to be somewhat distended. His liver was palpable 6 cm. below the costal margin and had a round nontender edge. There was no rigidity of the abdominal wall. The only palpable mass in his abdomen was his spleen which was felt at the level of the umbilicus and was nontender. There was moderate generalized enlargement of his lymph nodes. His hemoglobin was 11.5; his erythrocyte count was 3,150,000; his leukocyte count was 6,600 with 76 percent neutrophils, 4 percent eosinophils, and 20 percent lymphocytes. Gallbladder visualization was attempted, but the dye failed to concentrate in the gallbladder. A roentgenogram of the chest showed no change in the lesions of the ribs but the pleural reaction had cleared.

The patient's abdomen became more distended and he began to complain of pain in the right upper abdominal quadrant. A paralytic ileus developed which was relieved by a Wangensteen suction apparatus. Ascites developed after the ileus had receded. A paracentesis was performed and 1,900 cc. of a yellow, cloudy fluid was removed. An iliac marrow study was made and was reported as essentially normal. During the late phase of the illness the leukocyte count went up to 14,000 with an increase in the polymorphonucleocytes. The patient gradually became worse and on 7 September a yellow tint was noted in his scleras. Within 2 days this became an obvious jaundice. The icteric index rose to 67. The van den Bergh reaction was 4 plus. The quantitative bilirubin was 8.2. The cephalin-cholesterol flocculation test was 4 plus. At one time during this phase there was a strong suggestion of a palpable gallbladder. Ascites continued and frequent paracentesis was necessary. He died on 22 September.

The final clinical diagnosis was carcinoma of the gallbladder or liver. Autopsy revealed an adenocarcinoma of the liver, biliary-duct type with metastases in the lungs, adrenals, regional lymph nodes, pituitary, and bones (skull and ribs).

SUMMARY

This case is presented because the metastases to the skull and ribs resembled the lesions seen in multiple myeloma. None of the other cases that were reviewed showed these findings.

EDITOR'S NOTE: A paper entitled "Primary malignant disease of the liver" by C. H. Sanford, appears on pp. 304-312 of the August 1952 issue of the Annals of Internal Medicine.

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BOOK REVIEW

The Treatment of Injuries to the Nervous System, by *Donald Munro,* M. D., F. A. C. S., Surgeon-in-Chief, Department of Neurosurgery, Boston City Hospital; Associate Professor of Neurosurgery, Boston University School of Medicine; Assistant Professor of Neurosurgery, Harvard University Medical School. 284 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., publisher, 1952.

The author draws on his extensive clinical experiences in traumatic neurosurgery at the Boston City Hospital to combine in this compact, readable text both a philosophy of treatment and a practical guide to definitive treatment of the various injuries to the central and, to a lesser extent, the peripheral nervous system. Perhaps justifiably, because this volume is directed primarily to the general practitioner, the approach is quite dogmatic but, as the author states, the method has the virtues of practicality, the authority of evolutionary development over a period of years, and the honesty which is inherent in a constant checking of mistakes by the house staff and by postmortem examinations made by or under the direction of a medical examiner. The approach emphasized in this manual can best be summarized by quoting: "Even these attributes (enthusiasm, patience, attention to minutiae of therapy) are useless unless this surgeon is also convinced that no matter how extensive the paralysis * * * (the patient) ambulation with infallible 24-hour control of bladder and bowel * * * is well within the possibility of present-day treatment."

The general care of the patient from the time of injury, including transportation, necessary definitive operation, and rehabilitation in the convalescent stage, is emphasized throughout the manual. Equipment and technic are outlined for everything from a spinal tap to such craniocerebral operations as subtemporal decompression. There is a stimulating chapter on the cost of medical care in paraplegia and how it may be modified by rehabilitation services based on data acquired by a large insurance carrier of workmen's compensation liability. The last two chapters emphasize the psychology of rehabilitation and the important part played by the hospital personnel, from the trustees down, and the appropriate attitude to be fostered in the patient.—*Lt. Comdr. R. G. Berry, MC, U. S. N.*

COMPLICATIONS OF MUMPS

HARRIS D. RILEY, JR., *Captain, U. S. A. F. (MC)* (1)

ALTHOUGH usually considered a disease of childhood, many nonimmune adults contract mumps and in a high percent of patients it is followed by disabling complications. The incidence of mumps among military personnel is relatively high. Parran (2) stated that "next to the venereal diseases, mumps is the most disabling of the acute infections among troops." Wesselhoeft (3, 4) reported that 3,884,147 man-days were lost in World War I because of mumps. The fact that complications of mumps are much more common in adults than in children is important in military medicine.

CASE REPORTS

Case 1. Mumps with orchitis. A 22-year-old man was admitted to this hospital on 21 December 1951 with slight pain at the angle of both jaws of 4 hours' duration. He had never had mumps and there was no known exposure. Physical examination revealed a slight swelling at the angle of both jaws, and prominent posterior cervical nodes bilaterally. There was no redness or swelling of the orifices of Stensen's duct. The genitalia were normal. On the third day the patient developed tenderness and pain of both testes with low grade fever. This was treated supportively and subsided in 4 days. The patient was discharged on the seventeenth day.

Case 2. Mumps with pancreatitis. A 22-year-old airman was admitted to this hospital on 9 January 1952 because of swelling at the angle of the right jaw of 24 hours' duration. Another airman living in the same barracks had been hospitalized 2 weeks previously with mumps. This patient had never had mumps. There was marked swelling of both parotid glands with edema and redness of the orifices of Stensen's duct. The parotid swelling gradually subsided but on the seventh day the patient developed nausea without vomiting, cramping pain in the right upper abdominal quadrant and epigastrium, and a temperature of 104° F. There was slight tenderness in the right upper abdominal quadrant but no spasm. Serum amylase drawn at this time was reported as 229 units

(1) U. S. Air Force Hospital, Sewart Air Force Base, Tenn.

(2) Parran, T.: Health and medical preparedness. J. A. M. A. 115: 49-51, July 6, 1940.

(3) Wesselhoeft, C.: Mumps: Its Glandular and Neurological Manifestations. Virus and Rickettsial Diseases. Harvard University Press, Cambridge, Mass., 1940. p. 309.

(4) Wesselhoeft, C.: Medical progress; mumps. New England J. Med. 226: 530-534, Mar. 26, 1942.

per 100 cc. This patient was treated expectantly with analgesics and fluids parenterally. In 3 days he was afebrile and asymptomatic. He was discharged on the thirteenth hospital day.

Case 3. Mastitis following mumps. A 9-year-old girl was seen in the pediatric clinic of this hospital because of tenderness and swelling of both breasts. About 2 weeks previously she had developed bilateral parotid swelling following intimate exposure to a playmate with mumps. The swelling had lasted 1 week. Five days after it subsided she developed pain and tenderness, followed by slight swelling, of both breasts. There was no history of any predisposing cause of mastitis, and she had not had mastitis or mumps in the past. There was no parotid swelling and positive physical findings were limited to the breasts, which were exquisitely tender, slightly warm, and swollen. The changes were more marked on the left side. The breasts were covered with loose fitting sterile gauze and the patient was given analgesics as necessary. In 4 days all signs of inflammation had completely disappeared.

Case 4. Mumps meningo-encephalitis. A 20-year-old man was admitted to this hospital on 12 January 1952 because of headache. One week previously he had developed parotid swelling and pain which had gradually subsided in 5 or 6 days. He stated that he had had an "enlarged head" as long as he could remember. Two years previously, following a dental extraction, he had developed a left hemiparesis which had persisted. He had not had mumps. Physical examination on admission revealed a small, thin, young man who had an obviously enlarged head which measured 65 cm. in circumference. He refused to walk and complained of headache. A slight residual parotid swelling was present. There was no papilledema but there was evidence of a right facial nerve palsy. The neck was moderately rigid but Kernig's and Brudzinski's signs were negative. There was a residual left spastic hemiparesis. Lumbar puncture revealed slightly increased pressure, 14 mononuclear cells per cc., a positive Pandy's test, and a total protein of 213 mg. per 100 cc. Roentgenograms of the skull revealed hydrocephalic changes with flattening of the sella turcica but no erosion. Following the lumbar puncture the patient felt better and there was gradual disappearance of the nuchal rigidity and headache. A lumbar puncture, 9 days later, revealed only 3 cells and a decrease in the protein to 50 mg. per 100 cc. I believe that this patient had mumps meningo-encephalitis superimposed on a preexisting neurologic disease. Because this patient was a member of another service he was transferred to an Army hospital for investigation of his long-standing difficulties after treatment of his acute process.

Case 5. Recurrent mumps. A 25-year-old man was admitted to this hospital on 22 October 1951 because of pain in the right side of his neck and left side of his face of 2 days' duration. Soon after the onset of pain, swelling developed in his left parotid and right submaxillary areas. He had had mumps in 1946 and in 1950. Communication with the physician who treated him in 1950 confirmed the diagnosis. Physical examination revealed swelling of the left parotid and right submandibular salivary glands. The patient was afebrile. During his course in the hospital, with general supportive measures, the swelling of the affected glands gradually subsided and he was discharged on the tenth hospital day.

TABLE 1. *Incidence of mumps complications in the Willard Parker Hospital, New York, N. Y., from 1929 to 1932*

	Number of patients
Hospitalized with mumps	252
Orchitis	27
Pancreatitis	13
Meningo-encephalitis	2
Thyroiditis	1

DISCUSSION

The *incidence of complications* of mumps in the U. S. Army in World War II was 25 percent (5). Table 1 gives the analysis of the various complications as observed in a civilian communicable disease institution (6). *Orchitis* occurs in about 1 percent of children with mumps but in adults the incidence has been variably reported as from 10 to 50 percent (average 18 percent). The onset usually occurs between the fifth and tenth day after the swelling of the affected testis, and is accompanied by general malaise and discomfort in the inguinal region. The swelling progresses for 2 or 3 days and then regresses, usually returning to normal in from 7 to 10 days, but the course may be more severe. The testis is usually more severely affected than the epididymis. Occasionally, orchitis may be the only manifestation of mumps. Atrophy of the testis is common but impotence and sterility are rare.

Meningo-encephalitis is much more common in children but does occur in adults. Some investigators believe that a spinal fluid

(5) Stokes, J., Jr.: Mumps (epidemic parotitis). In Nelson, W. E. (editor): Mitchell-Nelson Textbook of Pediatrics. 5th edition. Philadelphia, Pa., 1950. pp. 620-624.
(6) Eagles, A. Y.: Analysis of 4 year epidemic of mumps. Arch. Int. Med. 80: 374-387, Sept. 1947.

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pleocytosis occurs in all patients with mumps whether signs of meningo-encephalitis are clinically present or not. In a series of 40 patients, routine examination of the cerebrospinal fluid revealed 16 with definite pleocytosis. In 6 of these there were no clinical signs of involvement of the nervous system *(7)*. Meningo-cephalitis may occur prior to, during, or following the phase of salivary gland swelling, although most cases appear from 3 to 10 days after the onset of the parotitis.

Pancreatitis is a comparatively rare complication of mumps. Radin *(8)* reported that out of almost 6,000 cases of mumps in military personnel, 14 patients had symptoms referable to pancreatitis. It occurs in both sexes and may be transient, mild, or severe. The onset is usually between the fifth and eleventh days with nausea, anorexia, and a high fever which returns to normal in from 3 to 5 days. Vomiting may or may not occur. Pain in the upper abdomen is usually intense (most frequently in the left upper quadrant) and is accompanied by tenderness. Constipation is the rule. Oophoritis in female and occasionally prostatitis in male patients may have to be ruled out *(9)*. Sailer *(10)* found jaundice in 4 out of 13 patients with this type of pancreatitis. High serum amylase values may be expected during the active stage of mumps and whenever there is obstruction to the normal flow of secretion from diastase-producing glands, particularly the parotid and pancreas *(11)*. The demonstration of a normal value of serum amylase in a patient with facial swelling is good evidence against mumps because 95 percent of patients with mumps have an elevated serum amylase *(12)*. Acute pancreatitis also produces elevation of the serum amylase. In pancreatitis due to mumps the serum amylase elevation is greater than the average expected levels of those in uncomplicated parotitis *(11, 12)*. The serum lipase is usually elevated in mumps pancreatitis and this may be helpful in making the diagnosis although clinical evidence is the best means of diagnosis *(13)*. The prognosis of this complication is good but the patient may be extremely ill during the course. There are only 2 reported deaths attributed to pancreatitis in which autopsy findings are given *(9)*.

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(7) Finkelstein, H. (West Orange, N. J.): Meningo-encephalitis in mumps. J. A. M. A. 111: 17-19, July 2, 1938.

(8) Radin, M. J.: Epidemic of mumps at Camp Wheeler. Arch. Int. Med. 22: 354-369, Sept. 1918.

(9) Brahdy, M. B., and Scheffer, I. H.: Pancreatitis complicating mumps. Am. J. M. Sc. 181: 255-260, Feb. 1931.

(10) Sailer, J.: Mumps. M. Clin. North America 3: 1423-1435, Mar. 1920.

(11) Zelman, S.: Blood diastase values in mumps and mumps pancreatitis. Am. J. M. Sc. 207: 461-464, Apr. 1944.

(12) Applebaum, I. L.: Serum amylase in mumps. Ann. Int. Med. 21: 35-43, July 1944.

(13) Candel, S., and Wheelock, M. C.: Serum amylase and serum lipase in mumps. Ann. Int. Med. 25: 88-96, July 1946.

Mastitis is a rare complication of mumps. It is characterized by pain, tenderness, and swelling of one or both mammary glands and may occur at any time after the appearance of the parotitis, although it is seen most commonly after the parotid swelling has started to subside. Mastitis may occur in either sex. The prognosis is good and clearing is usually complete.

The *protection* offered by an attack of mumps is usually lasting. However, there are interesting reports describing relapses and recurrences. A French soldier was reported to have suffered 6 attacks over a 3-year period, each attack corresponding to a separate exposure. In certain instances wind instrument players have experienced relapses and recurrences. Contrary to a popular notion, immunity following unilateral mumps is just as durable as when the swelling is bilateral *(14)*. Eagles *(6)* reported that 30 of 439 patients in an epidemic at Ft. Benning, Ga., gave a history of previous infection. Royce *(15)* presented evidence that recurrent swelling of the parotid gland may occur on an allergic basis. This may have been the cause in the patient here reported but as the serologic studies for mumps were incomplete and the patient is no longer at this installation, it is impossible to reach a definite conclusion.

The complications described are those most frequently associated with mumps but there are several others less commonly encountered. Oophoritis is said to occur in 5 percent of all women with mumps but is not as serious as orchitis in men because the ovary has no inelastic covering such as the tunica albuginea of the testis. Prostatitis, vulvovaginitis, dacryoadenitis, thyroiditis, myocarditis, pericarditis, and presternal edema have been reported. Deafness, labyrinthitis, facial nerve palsies, and optic neuritis may also occur but are extremely rare. Edema of the pharynx and larynx may occasionally necessitate tracheotomy *(3, 5, 14)*. Some workers have speculated on the possibility of the development of diabetes mellitus after mumps.

(14) Bradford, W. L.: Mumps (epidemic parotitis). In McQuarrie, I. (editor): Brenneman's Practice of Pediatrics. Vol. 2. W. F. Prior Co., Inc., Hagerstown, Md., 1949. pp. 1-9.

(15) Royce, S. W.: Recurrent swelling of parotid glands. Paper presented before meeting of American Pediatric Society at Old Point Comfort, Va., May 7, 1952.

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